JUN 8 1917
UNIV. OF MICH. LIBRARY

VOLUME 13 NUMBER 6

AMERICAN JOURNAL

OF

DISEASES OF CHILDREN

EDITORIAL BOARD

FRANK SPOONER CHURCHILL, Chicago
L. EMMETT HOLT, New York City DAVID M. COWIE, Ann Arbor, Mich.
JOHN LOVETT MORSE, Boston EDWIN E. GRAHAM, Philadelphia
JOHN HOWLAND, Baltimore

JUNE, 1917

PUBLISHED BY
AMERICAN MEDICAL ASSOCIATION
CHICAGO, ILL.

Entered as Second-Class Matter, January 4, 1911, at the Post-Office at Chicago, Illinois, Under Act of Congress of March 3, 1879

CONTENTS OF VOLUME XIII

JANUARY, 1917. NUMBER 1

PAGE

THREE CASES OF CONGENITAL CYANOSIS AFFECTING ALL CHILDREN IN A FAMILY AND A NEW METHOD OF DIAGNOSIS OF CONGENITAL HEART LESIONS. JULIUS H. HESS, M.D., AND ROY G. PEARCE, M.D., CHICAGO 1

THE PHENOL EXCRETION IN THE URINE OF INFANTS, INCLUDING THE NEW-BORN. CHARLES U. MOORE, A.B., M.D., M.Sc., PORTLAND, ORE. 15

STUDIES IN THE CHEMISTRY OF THE SPINAL FLUID OF CHILDREN. JEROME S. LEOPOLD, M.D., AND ADOLPH BERNHARD, B.S., NEW YORK 34

ALKALI AND EARTH ALKALI EQUILIBRIUM IN SPASMOPHILIA. CLIFFORD G. GRULEE, M.D., CHICAGO 44

ACROCEPHALY. PHILIP LEWIN, M.D., CHICAGO 61

THE EXCRETION OF SODIUM CHLORID SOLUTION INJECTED SUBCUTANEOUSLY. L. EMMETT HOLT, M.D., ANGELIA M. COURTNEY, AND HELEN L. FALES, NEW YORK 73

THE FACTOR OF INANITION IN THE DEVELOPMENT OF ACETONURIA IN CHILDREN. BORDEN S. VEEDER, M.D., AND MEREDITH R. JOHNSTON, M.D., ST. LOUIS 89

INFANTILE SCURVY. IV. THE THERAPEUTIC VALUE OF YEAST AND OF WHEAT EMBRYO. ALFRED F. HESS, M.D., NEW YORK 98

PROGRESS IN PEDIATRICS:

INDEX TO CURRENT PEDIATRIC LITERATURE 110

FEBRUARY, 1917. NUMBER 2

PAGE

EXPERIMENTAL STUDIES OF THE INTESTINAL FLORA. WARREN R. SISSON, M.D., BALTIMORE 117

THE HEMATOLOGY OF INFECTIOUS DIARRHEA. CHARLES J. BLOOM, B.Sc., M.D., NEW ORLEANS 128

THE SPECIFIC DIAGNOSIS OF TUBERCULOUS MENINGITIS. A PRELIMINARY COMMUNICATION. MICHIO KASAHARA, M.D., KYOTO, JAPAN 141

DIABETES MELLITUS IN CHILDREN. REPORT OF TWO CASES WITH A STUDY OF THE URINARY NITROGEN. P. C. JEANS, M.D., ST. LOUIS 145

SYPHILITIC DISEASE OF THE THYMUS IN INFANTS AND THE MODE OF ORIGIN OF THE DUBOIS ABSCESSES. JEAN OLIVER, M.D., SAN FRANCISCO 158

CLINICAL DEPARTMENT: CONGENITAL ATRESIA OF THE RIGHT AURICULO-VENTRICULAR ORIFICE WITH COMPLETE ABSENCE OF TRICUSPID VALVES. JULIUS H. HESS, M.D., CHICAGO 167

PROGRESS IN PEDIATRICS: RECENT WORK IN ANATOMY, PHYSIOLOGY AND PATHOLOGY OF CHILDHOOD. JAMES B. HOLMES, M.D., BALTIMORE 174

INDEX TO CURRENT PEDIATRIC LITERATURE 203

MARCH, 1917. NUMBER 3

PAGE

ACIDOSIS IN CHILDREN, WITH REFERENCE TO THE PATHOLOGIC ANATOMY. E. LACKNER, M.D., AND H. GAUSS, M.D., CHICAGO 209

THE OCCURRENCE OF ACIDOSIS WITH SEVERE DIARRHEA. O. M. SCHLOSS, M.D., AND R. E. STETSON, M.D., NEW YORK 218

FAMILIAL ICTERUS OF NEW-BORN INFANTS. I. A. ABT, M.D., CHICAGO 231

THE CALCIUM METABOLISM IN A CASE OF HEMOPHILIA. C. H. LAWS, M.D., AND D. M. COWIE, M.D., ANN ARBOR, MICH. 236

THE CEREBROSPINAL FLUID IN POLIOMYELITIS, WITH SPECIAL REFERENCE TO THE LANGE REACTION. P. C. JEANS, M.D., AND M. R. JOHNSTON, M.D., ST. LOUIS 239

THE SCHICK TEST IN POLIOMYELITIS, SCARLET FEVER, MEASLES, AND IN NORMAL CHILDREN. A. ZINGHER, M.D., NEW YORK 247

CLINICAL DEPARTMENT:

A CASE OF DIAPHRAGMATIC HERNIA. T. C. MCCLEAVE, M.D., OAKLAND, CALIF. 252

PROGRESS IN PEDIATRICS:

RÉSUMÉ OF WORK ON TUBERCULOSIS IN CHILDREN FOR 1916. MAY MICHAEL, M.D., CHICAGO 256

CURRENT PEDIATRIC LITERATURE 296

American Journal of Diseases of Children

VOL. 13 JUNE, 1917 No. 6

OCCLUSION OF THE AQUEDUCT OF SYLVIUS IN RELATION TO INTERNAL HYDROCEPHALUS *

MAX G. SCHLAPP, M.D.
Professor of Neuropathology, New York Post Graduate School
AND
BELDEN GERE, M.D.
Department of Pathology, Cornell University Medical College
NEW YORK

The object of this report is to present with all possible accuracy the results of the clinical, postmortem and histologic investigations in eight cases of internal hydrocephalus of the obstructive type, bringing special emphasis to bear on the obliterating lesion of the aqueduct of Sylvius as a cause of ventricular distention.

Of this group, examined without selection, all show either complete occlusion of the aqueductus cerebri or obliteration of the fourth ventricle through severe pathologic changes occurring in the ependymal or subependymal tissues of these regions.

The first four cases have already been shown before the New York Pathological Society[1] and the records reappear at this time in greater detail, together with the findings in four other cases not reported heretofore.

Beside the congenital, obstructive form of the disease as it appears in this review, a subdivision of this type of hydrocephalus occurs apparently idiopathically, in previously healthy young adults and older children, in which occlusion of the aqueduct may readily be made out in histologic sections. In such cases it is probable that a predisposing constitutional weakness has existed from the earliest time and that the ependymal cells have been stimulated by some local factors to excessive formative activity, so that gliosis, with consequent closure of the iter, takes place.

Although interference with the normal passage of cerebrospinal fluid, whether due to mechanical obstruction or to some pathologic change in the ependyma or more deeply lying structures of the aque-

* Submitted for publication Feb. 10, 1917.
* From the Department of Pathology, Cornell University Medical College.
—1. Proc. New York Path. Soc., 1911, **11**, 64.

duct, figures in the development of hydrocephalus, many cases have been recorded wherein no such factor can be made out. Similarly, the generally accepted idea of an obstruction to free communication between the ventricles, and between the ventricles and the general subarachnoid space, implying closure of the foramina of Magendie and Luschka, and in some cases of unilateral hydrocephalus, closure of the foramen of Monro, cannot be verified in pathologic studies of these conditions.

This variety of hydrocephalus offers an opportunity for further study of the origin, the removal, and hindrance to the normal movement of the cerebrospinal fluid.

The recently published studies of the cerebrospinal fluid by Weed and by Wegefarth, with an introduction by Cushing[2] consider this phase of the question in comprehensive manner, while the contributions of Frazier,[3] and of Frazier and Peet,[4] based on clinical and laboratory observation, have done much to enlighten us with respect to those problems which have long resisted solution.

Dandy and Blackfan[5] also in a report of their clinical and experimental work on hydrocephalus discuss the subject of the origin and disposition of the cerebrospinal fluid. Dixon and Halliburton[6] go into the points in question and give the results of their investigations dealing with the effects of extracts of the choroid and other glands on secretion of the choroid plexuses.

The surgical aspect of the subject has been well reviewed by Haynes,[7] who gives a detailed account of the physiology of intracranial pressure, discussing the modes of production and escape of fluid from the cavities of the brain.

Referring to the older literature on the subject, we find that L. W. Weber,[8] in summing up the pathologic considerations in a number of cases, came to the conclusion, not greatly at variance with present-day ideas, that three mechanical factors figure prominently in the production of hydrocephalus:

First, an increased fluid production due to inflammatory changes in the choroid plexuses, the ependyma and brain substance as well as obstructive processes in the choroidal vessels.

Second, impediment to the outflow of cerebrospinal fluid, via the venous and lymphatic channels, besides the obstacle of a frequently

2. Weed and Wegefarth: Jour. Med. Research, 1914, **31,** 21.
3. Frazier: Jour. Am. Med. Assn., 1915, **64,** 1119.
4. Frazier and Peet: Am. Jour. Physiol., 1914, **35,** 268.
5. Dandy and Blackfan: Jour. Am. Med. Assn., 1913, **61,** 2216.
6. Dixon and Halliburton: Jour. Physiol., 1914, **47,** 215.
7. Haynes: Ann. Surg., 1913, **57,** 449.
8. Weber, L. W.: Arch. f. Psychiat., Berlin, 1906, **41,** 61.

existing chronic, diffuse leptomeningitis, causing an impermeability of the subarachnoid spaces.

Third, lessened resistance of the ventricular walls due to inflammatory, degenerative or sclerotic processes in the affected areas. Weber believes that these three cardinal factors are operative in both acquired and congenital hydrocephalus, but that in the case of congenital disease the second element is present to a greater degree, while in the acquired form increased fluid production plays the more important rôle.

In the matter of diminished resistance of the walls of the ventricles in congenital hydrocephalus, Weber contracts the natural pliability of the fetal brain with the general or localized diseased condition of the brain substance in cases coming on later in life when dilatation may be symmetrical or confined to one, or to a portion of one, ventricle.

In that form of hydrocephalus characterized by hypersecretion, Gowers[9] has so frequently found meningitis associated with inflammatory changes in the ependyma and choroid plexuses that in his textbook he says, "the only known cause of hydrocephalus is meningitis."

Spiller and Allen,[10] in their report of two cases dealing with the causes of hydrocephalus, refer to Anton's support of Baginsky's opinion that acute hydrocephalus is identical with a nontuberculous inflammation involving not only the choroid plexuses, but also the walls and ependymal lining of the ventricles. Anton and Bonninghaus appear to be in accord with the opinion held by Gowers and others that these inflammatory changes are always associated with a simple or purulent meningitis.

Quincke[11] presents in detail the clinical studies of a number of cases in his effort to reach a definite understanding of the condition. Of the latter group of ten cases, two were examined post mortem, while in the others recovery, more or less complete, took place so that the clinical diagnoses could not be confirmed. In those cases which came to necropsy, no mention was made of the aqueduct of Sylvius. Beside the recognized causes of serous meningitis, Quincke points out the possibility of acute angioneurotic exudation from the choroid glands and ependyma, without fever or inflammation, a condition analogous to angioneurotic edema of the skin, and which he has designated angioneurotic hydrocephalus.

In summing up his conclusions regarding the pathologic changes in a number of cases of marked hydrocephalus in the adult, Parkes-Weber[12] says that "apparent thickening or slight granulation of the

9. Gowers: Diseases of the Nervous System.
10. Spiller and Allen: Med. Bull. Univ. Penn., 1907, p. 108.
11. Quincke: Samml. klin. Vortr., inn. Med., 1893, No. 23; ibid., Deutsch. Ztschr. f. Nervenh., 1897, **9**, 150.
12. Parkes-Weber: Brain, 1902, **25**.

ependyma should not be accepted as evidence of a genuine inflammatory process." Continuing, he recalls that "the whole ependyma has been subjected to chronic stretching and the pressure exerted by the effusion must of necessity damage the tissues immediately bordering it, and will probably cause a necrosis of individual cells, and, in chronic cases, a reactive change will follow and will be accompanied by a certain amount of cell infiltration, as it is wherever a local necrosis occurs." Parkes-Weber believes that Quincke's theory of the purely angioneurotic origin of some cases of hydrocephalus "lacks evidence to support it," but he advances the idea of extravasation into the brain cavities, comparing such collections of fluid to the serous effusions into the pleural and peritoneal cavities resulting from a serous pleuritis or peritonitis.

He explains the occurrence of idiopathic hydrocephalus as due in all probability to localized meningeal or ependymal inflammation producing an increase of cerebrospinal fluid. As an illustration of such localized inflammation, Parkes-Weber directs attention to the frequency, in fatal cases, of involvement of the posterior part of the roof of the fourth ventricle, and points out that the increase of fluid is due, not only to closure by the inflammatory exudate of the foramina in this situation, but that this region is one of the sites of election for the localized inflammation which leads to hydrocephalus.

This view is substantiated by Joslin's[13] report of eight cases of hydrocephalus following meningitis which showed this selective preference for the roof of the fourth ventricle. In all of his cases the foramen of Magendie and the openings into the cisterna lateralis had been closed and the roof of the ventricle rendered impervious by the inflammatory exudate. No changes were observed in the choroid plexuses and microscopic examination showed no thickening about the blood vessels. In one case of the series, that of a boy 16 years old, the aqueduct was occluded by a cystic tumor about 13 mm. in diameter, and adherent at several points to the choroid plexus of the third ventricle.

That the fourth ventricle is peculiarly liable to become the seat of a lesion causing dilatation of the cerebral ventricles and widening of the aqueduct of Sylvius, is abundantly attested by other workers, and cases of this type presenting symptoms of cerebellar tumor have been reported by Bramwell,[14] Spiller,[15] Finkelberg[16] and Rhein.[17]

13. Joslin: Am. Jour. Med. Sc., 1900, **120,** 444.
14. Bramwell: Brain, 1899, **22,** 66.
15. Spiller. Am. Jour. Med. Sc., 1902, **124,** 44.
16. Finkelberg: Deutsch. Ztschr. f. Nervenh., 1905, **29,** 135.
17. Rhein, J. H. W.: Jour. Am. Med. Assn., 1908, **51,** 1933.

Rhein's case, which showed the presence of a cyst in each cerebellopontine angle, is of further interest on account of an associated syringomyelia extending from the second cervical to the eighth dorsal segment of the cord. In the article Rhein says that Hinsdale, in a collection from the literature of 150 cases of syringomyelia, found hydrocephalus in fifteen.

Guthrie[18] says, in this connection, that primary hydrocephalus is frequently associated with syringomyelia, hydromyelia, and various forms of spina bifida, and these conditions he regards as being dependent on some abnormality of development of the meninges. Guthrie, in agreement with others, looks on meningitis as the principal etiologic factor in the majority of cases both of congenital and acquired hydrocephalus, and in his report of eight cases showing occlusion of the aqueduct, ascribes six to the results of meningitis, while one was due to the obliterating effect of a glioma of the pons, and, in the eighth, pressure of two symmetrical tubercular nodules effectually closed the iter.

The literature contains the accounts of a relatively small number of investigated cases of occlusion of the aqueduct, and the infrequency of careful and intimate examination in such instances is very much in evidence. Thus Bourneville and Noir cite a case of hydrocephalus in a child in which the aqueduct was completely closed, but a microscopic examination was not made. Touche, Oppenheim and D. Astros, without giving any specific instance, say that hydrocephalus affecting the third and lateral ventricles will follow plugging of the aqueduct of Sylvius, and von Becterew has observed obliteration of the aqueduct in some cases of cerebral syphilis with symptoms of dementia paralytica, but he does not go more into detail.

On the other hand, a considerable number of cases has been recorded in which hydrocephalus has been caused by obliteration of the fourth ventricle by papillary new growths springing from the floor, the roof and choroid plexus of the ventricle, notably the reports of Slaymaker and Elias,[19] Jacobson[20] and Douty.[21]

Spiller[15] reports a case of hydrocephalus internus presenting symptoms of cerebellar tumor due to closure of the aqueduct of Sylvius by proliferation of neuroglia, and in the same article he attributes a case of unilateral distention to partial blocking of the foramen of Monro from extension of an inflammatory process in the neighborhood. In another case of hydrocephalus which came under the observation of Spiller and Allen,[10] there was found occlusion of the aqueduct probably

18. Guthrie: Practitioner, London, 1910, **53,** 47.
19. Slaymaker and Elias: Arch. Int. Med., 1909, **3,** 289.
20. Jacobson: Hilton's Lectures on Pain and Rest.
21. Douty: Brain, 1885, **8.**

due to congenital malformation. The second of this group also seemed to be due to congenital malformation and was reported as a case of intense hydrocephalus without occlusion of the aqueduct.

The absence, in many instances, of demonstrable signs of a previous postbasic meningitis and the frequent development of a central gliosis, as demonstrated in this collection, lead to the belief that some form of obliterative process of the aqueduct, not necessarily associated with meningitis, could be discovered if diligently sought. As already suggested, the presence in the cerebrospinal fluid of some toxic product of metabolism or the influence of some disturbed function of internal secretion may be the exciting cause of a general or localized ependymitis leading, eventually, to closure of the aqueduct.

Thomas,[22] in his experimental work, has shown that hydrocephalus can be produced by injection into the ventricles of an irritating substance, and, using for the purpose a suspension of aleuronat, he has demonstrated that, through the acute inflammatory reaction which follows, obstruction may occur at the foramen of Monro, the foramen of Magendie, or in the aqueduct of Sylvius.

Accepting the well established facts regarding the origin and disposal of the ventricular fluid, it follows that, with plugging of the aqueduct, the essential cerebral avenues of escape are not sufficient to drain the ventricles and distention consequently results. With a definite obstruction to the normal flow through the aqueduct toward the fourth ventricle, facilities for the removal of cerebrospinal fluid are diminished, since the influence of the great cisterns and spinal subarachnoid cannot be brought to bear, thus throwing the burden of absorption on the cerebral sinuses and lymphatics.

REPORT OF CASES

Case 1.—*History.*—L. T., who was admitted to the Beth Israel Hospital, March 29, 1911, and who died the day following an exploratory operation, was a boy 3½ years old, the second of three children. Of the other children the eldest was living and apparently perfectly normal at the age of 9 years; the youngest was born dead after an instrumental delivery.

The patient, who had never been seriously ill, was born, the mother stated, with a large head, but that up to nine months prior to his death the child seemed to be perfectly normal in every other respect, exhibiting at all times good mentality. Nine months before his admission to the hospital the patient had a fall, striking on the head; this fall did not cause any external injury, but unconsciousness for some minutes' duration ensued, and this was quickly followed by vomiting. Six weeks later a well-defined and typical attack of epilepsy developed, and thereafter the child complained much of headache and suffered repeated attacks of convulsions, with unconsciousness. The last convulsive seizure occurred four months before his admission to the hospital; at that time and five months after his head injury, the child's mother noticed that his gait had become slow and uncertain and it soon became impossible for him to stand or walk without assistance. Vomiting which had no relation to the

22. Thomas: Jour. Exper. Med., 1914, **19.**

ingestion of food, increased in frequency and the boy became ill-tempered, execessively irritable and nervous. His mentality, however, remained clear.

Physical Examination.—The physical examination by Dr. Sachs showed that the patient was very well nourished, so much so, that the general and abundant distribution of fat suggested the possibility of some disturbance of the hypophysis.

The abdominal and thoracic organs showed no abnormalities. Neurologic examination demonstrated a spastic-ataxic gait, but there was no ataxia of the upper extremities and the Romberg sign was absent. The knee jerks were

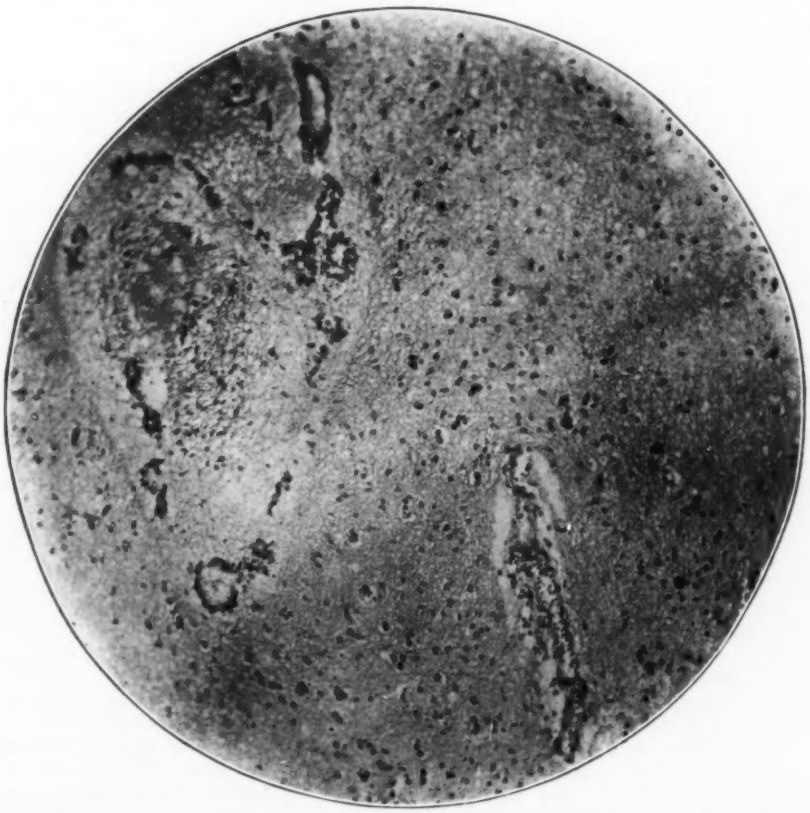

Fig. 1 (Case 1).—Showing central gliosis occluding the aqueduct of Sylvius, with proliferating ependymal cells arranging themselves in alveolar formation.

equal and not disturbed, but ankle clonus and the Babinsky reflex were present on both sides. Apparently there were no sensory disturbances. Examination of the cranial nerves showed double choked disk, with the left more swollen than the right; the retinal vessels were tortuous but there were no hemorrhages. Slight nystagmus appeared when the eyes were directed toward the right, but otherwise the movements of the eyeballs were not affected.

The pupillary reactions were not recorded.

Vision and hearing appeared to be normal in response to the usual tests. There was no deviation of the tongue when protruded, and the act of swallowing was performed perfectly. The sense of smell was not tested and the other cranial nerves showed nothing pathologic.

In the right parietal region a small angioma was exposed when the scalp was prepared for operation. In its greatest circumference the head measured 62 cm., the fontanels were closed and the frontal eminences were unusually prominent. The veins of the forehead were much distended.

Operation.—Decompression was performed by Dr. Sachs April 15, and the following day the patient died.

Gross and Microscopic Examination.—The gross specimens of the case submitted for examination were cut through the crura and complete occlusion of the aqueduct clearly shown.

Microscopic section made through the pons confirmed the macroscopic appearance and demonstrated the existence of a central gliosis, with a marked proliferation of ependymal cells, which in places near the central lesion showed a glandular arrangement tending to the formation of cysts.

In the subependymal glia tissue fibrils were present to a considerable depth and the same process could be observed extending ventrically along the raphé, and in this portion cellular elements were also abundant.

Examination of a portion of the cerebral cortex presented evidence of a general ependymitis, and section through the cortex, including a portion of the ependymal lining on one lateral ventricle, showed the existence of small nodules in which cell elements predominated.

The meninges showed no inflammatory process and no abnormality of the hypophysis could be made out. From these changes it is to be inferred that closure of the aqueduct of Sylvius had existed from the earliest period of development of the child's brain, and the fall sustained may perhaps be considered as an exciting cause of an ensuing ependymitis with consequent increase of the cerebrospinal fluid.

CASE 2.—*History.*—To Dr. John Howland, in whose service at Bellevue Hospital this case occurred, and to Dr. Charles Norris, pathologist, acknowledgment is made for the clinical history, the necropsy report and the pathologic specimens in the case of G. N., a boy, 5½ months old, admitted to the hospital March 18, 1911, and who died March 27, 1911.

It was said by the mother of this baby that she had noticed no abnormality of development until, during the second month of his life, her attention was directed to the increasing size of the baby's head. Shortly after this the child began to have peculiar spasms lasting about three minutes, during which the upper and lower extremities became very stiff. These convulsive attacks recurred three or four times a day. Later, when the patient was about 4 months old, a lumbar puncture was made. The removal of a little more than a pint of cerebrospinal fluid brought about a marked improvement, which continued for six weeks, when the child again grew gradually worse and died March 27, 1911.

The patient, who was the only child, was delivered at term with the aid of instruments, after an active labor of twenty hours' duration. The parents of this child were living and well and there was no family history pointing to any possible etiologic influence.

Physical Examination.—Physical Examination showed an abnormally developed, but fairly well-nourished infant with a high, protruding forehead, prominent parietal bosses, large open fontanels and the whole vertex soft and pulsating.

Measurements of the head were as follows:

	Cm.
Greatest circumference	63
Occipito-mental circumference	61
Diameters:	
Occipito-frontal	22
Occipito-mental	21
Biparietal	17
Bitemporal	13.5
Suboccipito-bregmatic	18
Frontal-mental	18

Chest measurements, at full expansion at the level of the nipples, 39 cm.; at the costal margin, 41 cm. Abdomen, at the level of the umbilicus, 37 cm.; length of body, 68 cm.

The skin was rather mottled in appearance and showed a few erythematous areas. Examination of the thoracic and abdominal organs was negative.

On admission to the hospital the temperature was 99.6 F. and remained practically normal until the end; the day before death occurred the temperature fell to 94.8 and then rose again to normal. The limbs were somewhat spastic and the knee jerks were exaggerated.

Fig 2 (Case 2).—Lesion of central gray matter showing closure of the aqueduct through a distinct proliferation of glia cells, giving to the new formation the appearance of a glioma.

Necropsy.—The necropsy confirmed the head measurements taken before death, and, in addition, records the dimensions of the anterior fontanel as 17 cm. in length; 15 cm. in its greatest width. The right parietal bone was considerably ossified and more prominent than the left. The tissues of the scalp were thin through distention, and, on removal of the cranial bones, the brain was revealed as a large sac filled with a clear fluid.

The cerebral cortex measuring 1 mm. in thickness was entirely without fissures or convolutions. The basilar ganglia appeared normal, the caudate nucleus on each side being especially well formed. The olfactory lobes and optic tracts were remarkably flattened and the hypophysis cerebri appeared softened.

The cerebellum, the medulla and the nerves at the base of the brain showed no abnormalities and the vessels in these regions were normal, while evidences of meningitis were absent.

Microscopic Examination.—Sections made through the midbrain showed complete occlusion of the aqueduct of Sylvius and microscopically the obliteration was seen to have been brought about by proliferation of glia cells, which, with a great increase of glia fibrils, had practically erased the contour of the central gray matter, producing the appearance of a glioma. The disposition of the ependyma cells to arrange themselves in the form of cysts was pronounced.

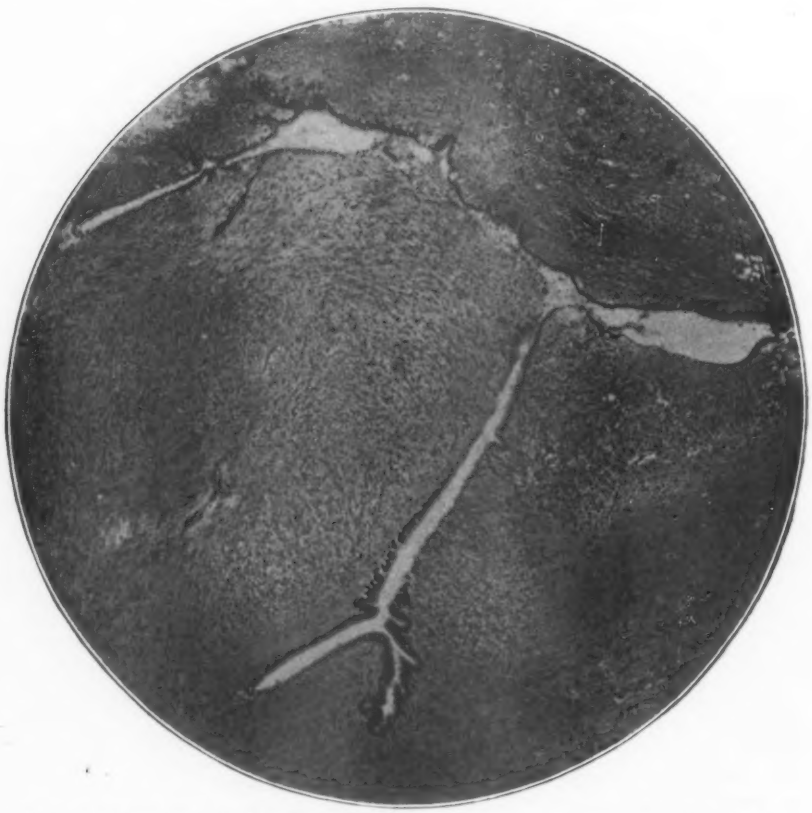

Fig. 3 (Case 2).—Another section of central gray matter showing obstruction by the new formation.

Above the point of obstruction the aqueduct became patent and appeared much wider than under normal conditions, and sacculated, while the immediately subjacent structures had been invaded by the same process.

The meninges appeared normal and the choroid plexuses presented no demonstrable changes.

CASE 3.—*History.*—This patient, a girl, 2½ years old, was admitted to the children's ward at Bellevue Hospital, March 28, 1905, with the statement of the mother that the child was born with a very large head and that the condition had been gradually increasing. Of the family history little was learned beyond the fact that the parents were living and well, and that of several brothers and sisters all were perfectly normal.

Physical Examination.—It appeared that the child took its nourishment without any trouble, but had never been able to move her arms or legs. At the hospital it was found that, aside from complete paralysis of the extremities, the child, as far as could be determined, reacted to painful sensory stimuli by crying. The child was blind and did not seem to hear, but all the movements of the face were carried out in perfect coordination as observed when the patient cried, or at times seemed to smile. This showed that coordinated movements of the facial muscles were possible through emotional stimulation.

In posture this patient lay with the head fully extended, pressure at the point of contact between the back and occiput causing an erosion of the skin. The eyes could not be closed on account of the tension of the skin which drew the eyelids upward. Any movements of the patient's head or extremities caused her great distress.

The temperature of the patient was normal until two weeks before death, when it reached an elevation of 103 F., and during the last week of her life it remained above 101, dropping at the last to 97.

The extremities were spastic, legs and feet extended; the arms were flexed at the elbows and fingers folded in on the palms. In the upper extremities marked tremor was present. The tendon reflexes were very active.

Fig. 4 (Case 3).—Gross specimen showing greatly distended third and lateral ventricles, with thin cerebral cortex. The basal ganglia and entrance to the aqueduct are clearly pictured.

The pupils were moderately dilated, equal and insensitive to light. There were no ocular paralyses. There was some rigidity of the abdominal muscles, but otherwise examination of the thoracic and abdominal organs was negative.

Necropsy.—Necropsy was performed May 10 by Dr. Charles Norris, and to him ackuowledgment is made for the pathologic material. In general, the postmortem examination showed no special deviations from the normal, the findings being negative except for the presence in the posterior portions of the lower lobes of both lungs of hypostatic congestion and a cystic condition of the left suprarenal gland.

Head: Parietal, frontal and occipital bones were found to be about the thickness of heavy parchment. The measurements were as follows:

Posterior fontanel, small but open.	
Anterior fontanel, along the coronal suture	22 cm.
In direction of sagittal suture	7 cm.
Lamboid suture, closed.	
Measurements of face:	
Chin to nasion	10 cm.
Malar to malar	9 cm.

In its greatest circumference the head measured, over the parietal and occipital protuberances, 67 cm., and the greatest anteroposterior diameter from the nasion to the external occipital protuberance was 52 cm.

On opening the calvarium the dura was found to be adherent, thin and translucent. There was no distinct evidence of pacchyonian granulations, and the superior longitudinal sinus was entirely empty.

After carefully removing the dura, the cortex appeared smooth and bloodless, with no suggestion of fissures or convolutions. The posterior third of both hemispheres consisted of a very thin, transparent membrane through which could be seen the large cavities of the distended lateral ventricles. The cortex of the anterior two thirds of both hemispheres was very thin, measuring 2 mm. in thickness, but gradually increasing toward the base of the brain where it passes over the lenticular nuclei. Here the cortex measured from 5 mm. to 1 cm. in thickness.

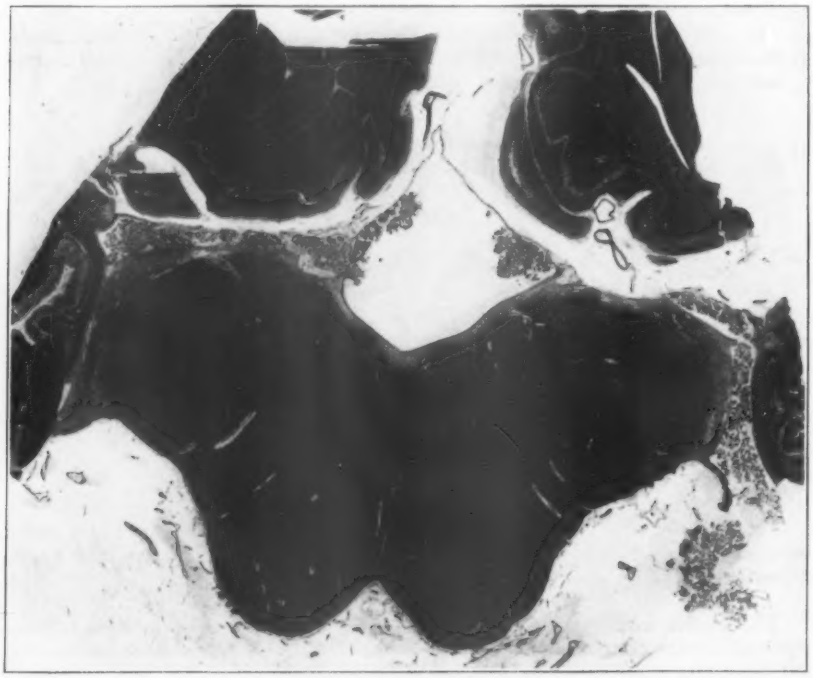

Fig. 5 (Case 3).—Cross section of medulla showing open fourth ventricle, entire absence of the pyramids, and choroid plexus unaffected.

The lateral ventricles were opened dorsally, liberating about 5 liters (estimated) of a clear, light-colored fluid. The corpus callosum was missing.

The basilar ganglia were present, though somewhat diminished in size. Lying between the optic thalami, the third ventricle appeared as a depression, and leading from its anterior portion the distended infundibulum could be made out, surrounded only by a thin membrane with the normal nervous tissue missing.

From the posterior portion of the third ventricle there might be seen the entrance to the aqueduct of Sylvius which later sectioning showed to be completely occluded. The fourth ventricle was not distended.

The optic nerves were smaller than normal, but were not compressed as were the olfactory lobes and olfactory tracts; all of the other cranial nerves appeared

to be somewhat small, but otherwise showed nothing pathologic. The crura and pons from the ventral surface appeared normal, while the medulla and spinal cord showed plainly the entire absence of the pyramidal tracts. The cerebellum and peduncles were normal. The accompanying photographs (Figs. 4, 5 and 6) clearly demonstrate the absence of the pyramidal tracts, and this explains at once the complete paralysis of the extremities. But how are we to account for the coordinated movements of the facial muscles, the motor neurons being absent?

It is well known that, as we trace the phylogenetic development of the central nervous system from the lower to the higher forms of animal life, the function of motion is transferred from the basal ganglia to the cerebral cortex. If, for instance, the motor cortex of a dog or cat be destroyed, it will be found that these animals, after the shock of the operation has subsided, are still capable of walking, although in a disturbed manner, showing that some center other than the motor center in the cerebral cortex still has an influence over movements of the extremities. This center is undoubtedly situated somewhere in the basal ganglia.

Fig. 6 (Case 3).—Section of spinal cord in the dorsal region showing again that the motor tracts are missing. The gray matter is normal.

Removal or destruction of the motor cortex of the human brain or brain of the higher monkeys, however, results in complete paralysis of the extremities, a fact which shows that the motor function has been completelty transferred from the basal ganglia to the cerebral cortex. It seems, however, that this transfer of the motor function has as yet not taken place completely in movements of the face, and particularly has it not taken in those muscular actions expressing the emotions; so we must conclude that the emotional movements of the face are still, to a certain extent, vested in the phylogenetic earlier motor centers of the basal ganglia. This conclusion is amply justified by the total absence, in Case 3, of a cerebral motor cortex.

Histologically, the essential features of gliosis are present in the pathologic changes which closed the aqueduct in this case, and while the process does not appear to be as active and aggressive as in the preceding pictures, occlusion is complete. Above the lesion the aqueduct is much dilated and the ependymal lining is normal. Sections show clearly the absence of the pyramidal tracts at all levels of the cerebrospinal axis.

The meninges did not show the presence of any inflammatory exudate and there were no appreciable changes in the choroid plexuses.

CASE 4.—*History.*—This child was admitted to the Presbyterian Hospital, Oct. 20, 1909, at the age of 6 months, and came immediately under the observation of Dr. John A. Hartwell, whose interest eventually made possible a histologic study of this case.

According to the records of the hospital, the child was born normally at full term, of perfectly healthy parents. Of the other children in the family, two brothers were living and well at the ages of 12 and 9 years, respectively; each of two sisters died within one hour after birth, the delivery in each case having been instrumental.

This baby nursed up to the time of her admission to the hospital. When 6 weeks old, however, she suffered from serious bowel trouble, a condition which was described by the family physician as marasmus. The illness continued about six weeks and the child had "weak spells"—"internal convulsions," the mother called them—when she seemed dazed and almost unconscious for a whole day at a time. At the age of 3 months the child had a well-defined convulsion, lasting about five minutes; this was the only pronounced epileptiform attack which occurred, and was caused, the physician believed, by a condition of chronic constipation which required the daily use of a cathartic.

The mother stated that the child's head had been large since birth and to her had never seemed like other children. She had never attempted to raise the head, and one eye had turned outward from birth. The baby's eyesight during the last month had been very poor, and the mother believed that the baby could not see at all, but lately she had appeared to recognize her mother's voice. During the previous six weeks the head had grown considerably in size.

Physical Examination (by Dr. Davis).—The patient was a moderately well-nourished child with an enlarged, square head.

Measurements:

	Inches
Occipito-frontal	19½
Occipito-bregmatic	18¾
Occipito-mental	19¾
Fronto-mental	19¾
Mastoid to mastoid over the head	15½

The anterior fontanel was open and its anteroposterior diameter and the lateral diameter each measured 4 inches. The parietofrontal sutures had not yet united and there was no pulsation.

Eyes, ears, nose, mouth and face were of normal size. There were no glandular enlargements to be made out. Examination of the thoracic and abdominal organs was negative. The knee jerks were exaggerated and there was ankle clonus and the Babinsky reflex on both sides.

Operation and Course.—Oct. 22, 1909, an operation was undertaken by Dr. Hartwell for the purpose of draining the ventricles of the brain into the peritoneal cavity.

Procedure: Three incisions were made: first, in the right side of the abdomen and through the peritoneum, about two inches below the costal margin; second, over the middle of the right clavicle; third, 1 inch above and behind the right external auditory meatus. A probe was then passed upward subcutaneously from the abdominal incision to the clavicular incision. A grooved director was passed from this latter opening to the wound in the scalp, making a channel for the probe. A heavy silk thread was then drawn through these tracks, making a subcutaneous channel from the skull to the abdomen. A double twisted wire was then made to replace the silk. The dura was incised through a quarter-inch trephine opening in the skull and the end of the loop of

wire was driven by means of a probe into the brain in the direction of the opposite external meatus. The lower end of the wire, formed into a loop, was passed into the peritoneal cavity and the three wounds closed.

A considerable amount of cerebrospinal fluid was lost at the time of the operation, but the patient's condition was satisfactory, and on October 30 she left the hospital improved.

The temperature following the operation ran along at 102 F. or over, 104 being the highest temperature recorded. On the sixth day the temperature began to fall and then remained below 100. The pulse varied from 140 to 200 per minute and slowed finally to 110 to 100; respirations from 40 to 30. On the fifth day following the operation the sutures were removed and the wounds

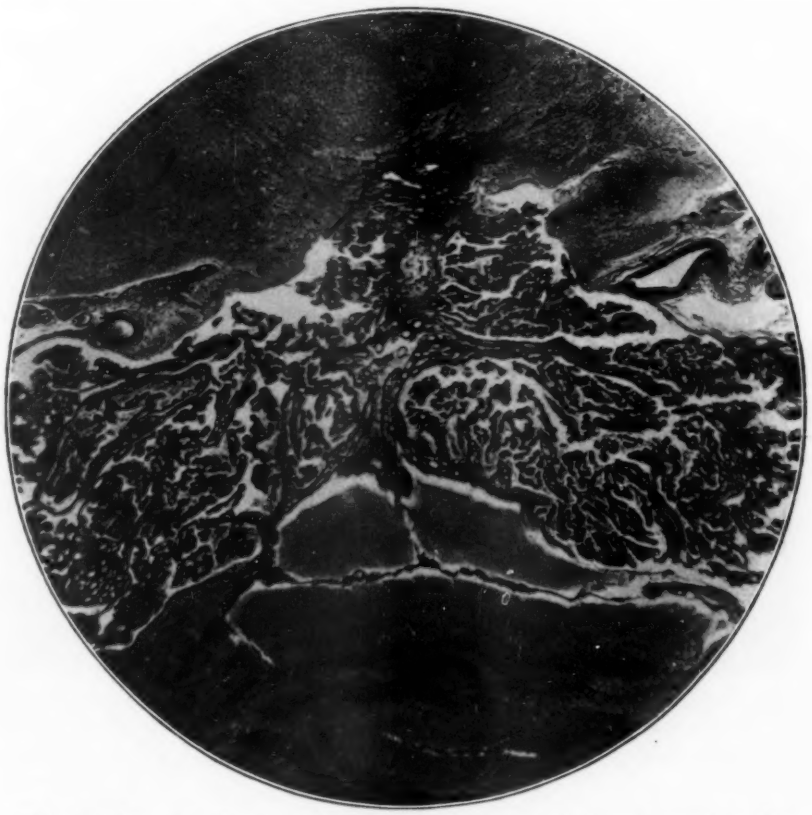

Fig. 7 (Case 4).—Section taken at the beginning of the fourth ventricle, showing the first appearance of the obliterating process as a pedunculated mass composed of proliferating choroidal cells. Except near the choroid plexus, it shows no special disturbance.

were found to be clear. In spite of the rapid pulse it always maintained fair quality. The patient had numerous muscular twitchings bilaterally for several days, but these gradually lessened and finally disappeared. The fontanels became very much depressed, showing that drainage from the ventricles was actively taking place.

The child remained with the parents until Jan. 4, 1911, when she was admitted to Bellevue Hospital suffering with mastoiditis complicating her hydro-

cephalus, which had not greatly improved during the interval. The baby's general condition was good and the neurologic examination gave about the same results as at first observed. The fontanels were closed. There was slight internal strabismus, and while the pupils reacted sluggishly to light, it was evident that the child was blind.

The mastoid condition was relieved by operation, and at the same time Dr. Hartwell removed the silver wire which, in the first instance, had been introduced for purposes of drainage of cerebrospinal fluid. It was found that the subcutaneous channel was completely lined by epithelium and permanent drainage into the peritoneal cavity established. Improvement was manifest for a time after these aspirations, but death occurred in her own home a few weeks later.

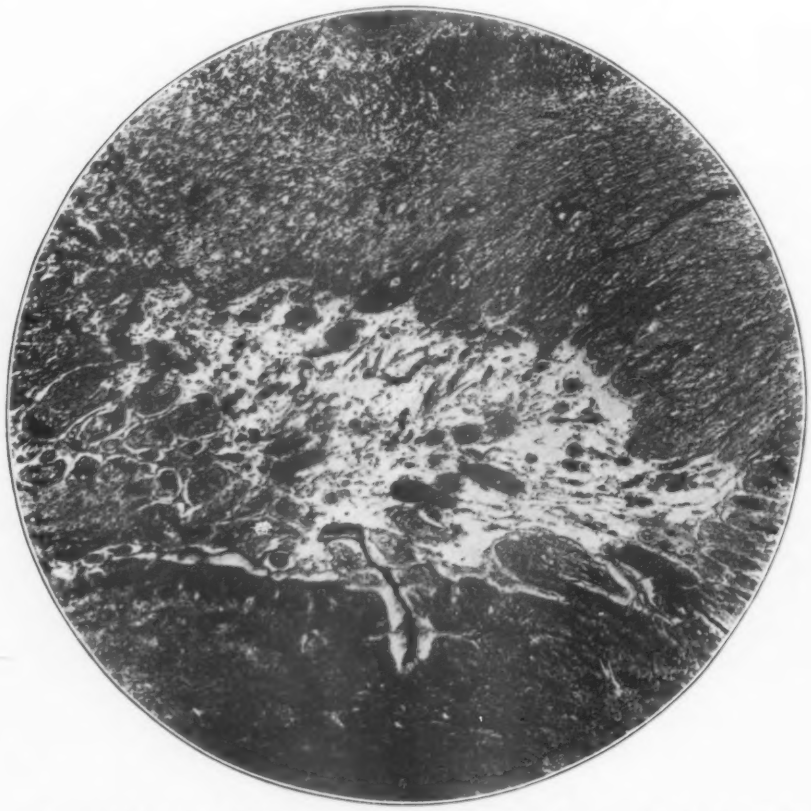

Fig. 8 (Case 4).—Section greatly enlarged, showing at the level of the eighth cranial nerve invasion of the pons and cerebellum by the proliferating new growth, causing complete occlusion of the ventricle.

Microscopic Examination.—This shows complete obliteration of the fourth ventricle and the aqueduct of Sylvius.

In Figure 7 may be seen the process beginning in the raphé of the floor of the ventricle, appearing as a pedunculated tumor mass composed of choroidal cells, with invasion of both the cerebellum and medulla. Passing cephalically, these choroidal cells increased in quantity, and in Figure 8, at the level of the nucleus of the eighth cranial nerve, the ventricle appears to be completely filled.

Here as at other levels proliferating choroidal cells invade the nerve tissue of the cerebellum, medulla and pons in typical neoplastic fashion.

The aqueduct of Sylvius throughout its course is entirely closed, not by choroidal cells, but by proliferation of the surrounding glia cells.

At those points where the cerebellum and medulla have been invaded, choroidal cells, having taken on the characteristics of ependymal cells, have arranged themselves in the form of small cysts.

The choroidal cells are unquestionably spongioblastic, derived from the same mother cell, which gives origin to the glia and ependymal cell.

CASE 5.—*History.*—The only clinical history obtainable in this case is taken from the records of the City Hospital and from the nurses' bedside notes, and relates principally to the surgical treatment which this child of 15 months received from the time of his admission to the ward, Feb. 11, 1911, to the date of his death, Oct. 26, 1911.

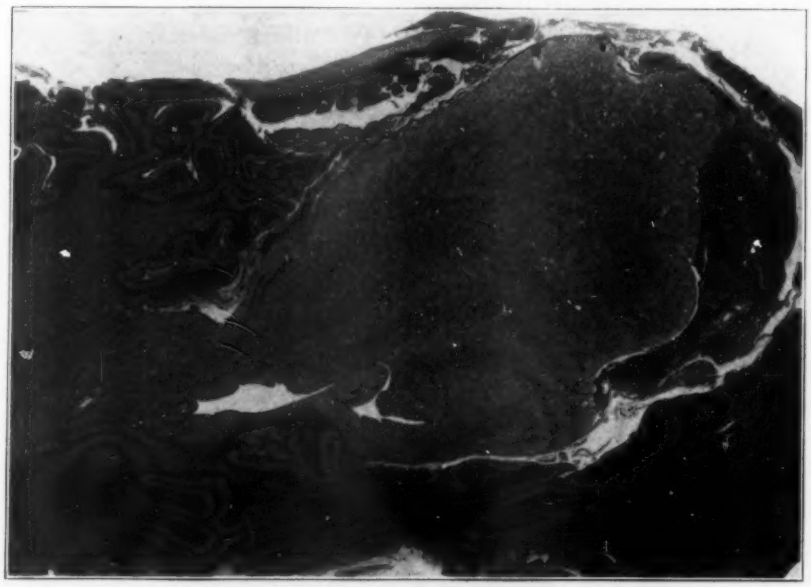

Fig. 9 (Case 5).—Cross section in the region of the nucleus of the third cranial nerve showing presence of small tumor mass, histologically a glioma, proceeding from the floor of the sylvian aqueduct and partly occluding the lumen.

The specimens of this and the following case were furnished by Dr. John H. Larkin, pathologist to the Department of Charities, New York City.

The case is that of a boy, 1 year and 3 months old, who showed on physical examination a large head, globular in form, with greatly enlarged fontanels, through which fluctuation could be plainly felt. The face was small and pinched in appearance, eyes prominent and bulging, and the superficial veins of the head distended.

Dr. Dawbarn, in whose service at the City Hospital the case occurred, attempted to drain the lateral ventricles into Stenson's duct, and with this end in view an operation was performed by him, Sept. 25, 1911.

Operation and Course.—The records state that a large semicurved needle was introduced into the anterior fontanel well away from the middle line and made

to merge from the anterior part of the parotid gland. A No. 18 silk tape was then carried through, cut off at each end even with the surface, and the ends buried by closing both incisions.

The greatest circumference of the head before operation was 67.5 cm. After operation, which was attended by a considerable loss of cerebrospinal fluid, measuremeht of the head showed a circumference of 65 cm., and by September 30 this was further reduced to 63.7 cm.

During the month of August it appears that the baby had four severe convulsions of from five to seven minutes' duration, and again on September 24, the day before the operation, a very severe convulsion occurred; and on September 25, shortly after the operation, there is a history of repeated slight attacks. Vomiting and unusual restlessness then set in and continued for two days, when the child again became quiet and retained most of his nourishment. It was noticed on September 27 that the patient's body and extremities had become very stiff and vomiting occurred more frequently. During the whole of his stay at the hospital the baby was, at times, extremely restless and frequently cried throughout entire day and could not be pacified.

Fig. 10 (Case 5).—Transection through pons, again illustrating effect of a small glioma with active proliferation of glia cells at the base of the new growth. Changes in the subependymal structures and in the choroid plexus may be observed under high magnification.

Gradually increasing weakness and frequent periods of restlessness marked the progress of the succeeding days, and on October 9 a second operation similar to the first was performed on the opposite side of the head. During the three days following this operation the child seemed better and was making satisfactory progress, when a marked weakness supervened, and, although the patient took nourishment fairly well, this weakness increased and gradually it became more and more difficult to administer nourishment. Death finally took place Oct. 26, 1911.

Examination of Brain.—The gross specimen in this case was of unusual appearance, the bases of the anterior crura being almost unrecognizable as

such. The right crus, very much enlarged, appeared as a tumor mass about 1.5 cm. long and 2 cm. in its transverse diameter; the left crus was smaller, measuring about 1 cm. in thickness.

These masses disappear at a point where the crura pass into the pons; cephalically, they extend to a point considerably above the red nuclei. Between these two masses the neutral fissure reaches between the red nuclei to the level of the aqueduct.

The aqueduct in the region of the nucleus of the third cranial nerve appears to be entirely closed, but microscopically a minute sacculated opening remains, and this is partly filled with a small tumor formation growing in from the roof

Fig. 11 (Case 6).—Syphilitic gumma proceeding from the pia-arachnoid, invading and partially occluding the fourth ventricle. Characteristic changes in the lymph and blood vessels are much in evidence.

of the aqueduct; similarly, at a point where the aqueduct normally tends to widen out centrally, another small growth makes its appearance, invading the lumen of the aqueduct from above. At this same level a very small new formation may be seen taking origin from the subependymal structures of the floor of the aqueduct.

Microscopic Examination.—Histologically, these invading masses are gliomata. The choroid plexuses do not appear to be the seat of inflammatory changes, but many choroidal vessels are engorged, indicating a lesion of obstructive type interfering with the normal functioning of these glands.

The general ependymia shows no abnormality, but in the more deeply lying tissues there is very evident abnormal cell activity, with extensive perivascular round cell infiltration. Here several contributory factors in the production of hydrocephalus may be seen in combination. Not only is the aqueduct encroached on by protrusion into its lumen of various small gliomata, but the processes of secretion and absorption have been disturbed through involvement of the choroidal vessels and ventricular walls.

CASE 6.—*History.*—As in the preceding case, the hospital records are incomplete, the available history dealing chiefly with incidents of the child's birth. The baby was born at full term of a young multipara, who, as far as could be

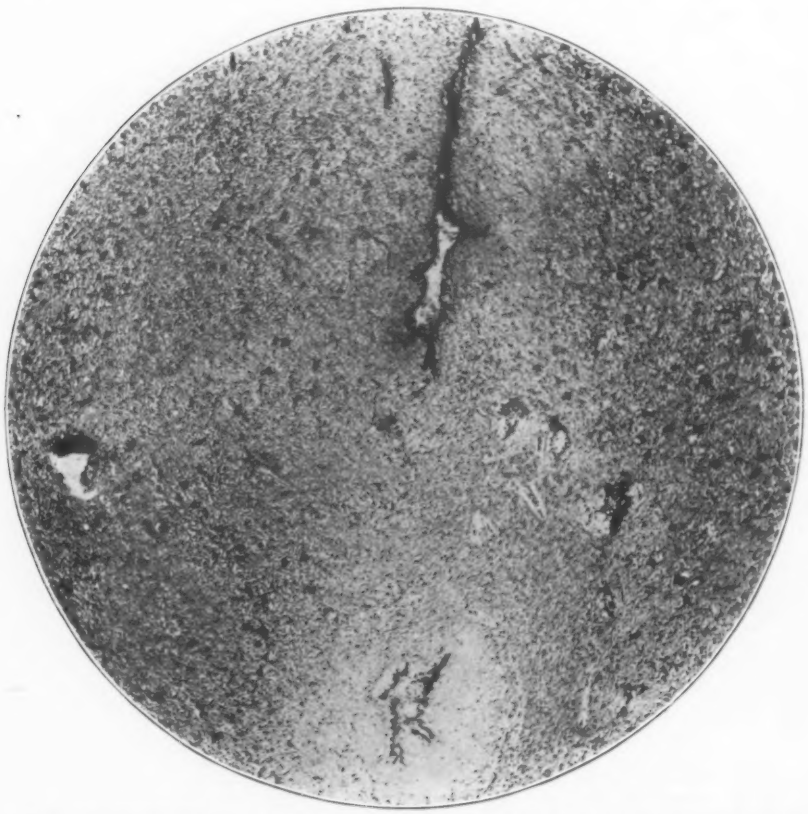

Fig. 12 (Case 6).—The region of the nucleus of the third cranial nerve, showing complete occlusion of the aqueduct and activity of the ependymal and perivascular changes in the subependymal tissue.

learned, was a perfectly normal individual. Her other children were born normally, and they made entirely satisfactory progress through infancy and childhood. In this instance, however, labor was prolonged and difficult, the second stage lasting thirty-six hours, and it was only after craniotomy had been performed that delivery could be accomplished. The mother made a good recovery and returned the next year to the maternity wards of the City Hospital, where she gave birth to a perfectly normal child.

While nothing was discovered in the history on the part of the mother to suggest a possible cause of the hydrocephalus, the fetal brain was examined

by the Noguchi method and *Spirochaeta pallida* were found in the cerebral tissue. This placed the origin of this case definitely on a syphilitic basis, and is the only one of the series to react in this manner.

Further microscopic evidence of the syphilitic nature of this lesion is to be found in high-power study of the sections.

Examination of Brain.—The gross specimens showed fairly well-developed pons and medulla, while the basal ganglia were of fair size and consistency. The cerebral cortex, however, was thinned out to a mere sac-like formation, containing very little brain substance and possessing no convolutions or fissures.

Microscopic Examination.—Microscopically, the picture is that of meningoencephalitis with characteristic blood-vessel changes in which the perivascular lymph spaces are crowded with infiltrated round cells and lymphocytes. The aqueduct in the sections is completetly closed and considerable cell activity is to be made out in the ependyma and periependymal tissues.

Many small spaces lined by cuboidal cells give the appearance of cysts, suggesting the tendency of certain modified ependymal cells under stimulation to arrange themselves in this manner, but undoubtedly these stand in relation to obliterated vessels remaining as evidence of earlier lymph stasis with consequent changes in adjacent areas.

In the region of the nucleus of the eighth cranial nerve a small gumma encroaches on the fourth ventricle, impeding the flow of cerebrospinal fluid. This pedunculated mass, originating in the pia-arachnoid, stands in close association with the choroid plexus of the fourth ventricle, and while the plexus and the choroidal cells themselves show no changes, the inflammatory exudate extends laterally, following for a considerable distance the pia covering of the gland. The exudate itself, marked by the presence of many lymphocytes and polynuclear leukocytes and showing areas of necrosis, is the seat of an active as well as a chronic inflammation.

CASE 7.—*History.*—To Dr. Elise L'Esperance, pathologist to the New York Women's Infirmary and assistant in pathology at the Cornell University Medical College, we are indebted for the specimens and history of this case.

The history shows that the parents of this child were young and healthy, and that the birth occurred as a miscarriage in the seventh month of pregnancy. The child lived about one hour.

Necropsy.—At the necropsy, performed by Dr. James Ewing, the head presented a high grade of hydrocephalus, with all the ventricles dilated. Measurements, however, were not taken.

The convolutions were flattened and the cerebral cortex measured from one-eighth to one-fourth of an inch in thickness. The lungs were completely collapsed and the heart on the right side was hypertrophied.

Both kidneys were cystic, the left atrophic, measuring 1.5 cm. by 1 cm.; the right hypertrophied, measuring 7 by 4 by 3.5 cm. There were cysts in the dilated renal tubules. The suprarenals were not affected; thyroid normal. The spleen and gastro-intestinal tract showed no abnormalities.

Microscopic Examination.—To the naked eye the aqueduct appears to be closed, but microscopically a passage remains in the shape of a minute slit, about which an active proliferative process may be made out, while at either end of the opening there is a well-advanced gliosis. Inflammatory and vascular changes have occurred in the choroid plexuses, with lymphocytic infiltration of the vessels of the medulla, pons and cerebral cortex.

CASE 8.—*History.*—An infant, 7 months old at the time of her death, was born, so the mother declared, with a head of normal size, but labor was difficult and delivery was finally effected after the ordinary forceps operation.

On admission to the hospital, Oct. 9, 1913, the chief complaints, as entered in the clinical history, were abnormally large head, great weakness and listlessness.

During her earliest infancy the baby suffered from obstinate constipation, but otherwise was perfectly normal, and the increase in the size of the head was so gradual that the abnormality was not definitely recognized until, in the fifth month of life, a friend directed the mother's attention to the now apparent disproportionate size of the child's head.

The mother then placed the infant in the Babies' Hospital, where she remained under observation for a short time. It had been the mother's custom to return to the hospital daily for the purpose of nursing the baby, and on the occasion of her last visit she found the child in a weak and exhausted condition following a lumbar puncture, and against advice removed the patient to her own home. Later, on the same day, however, the mother applied for admission to

Fig. 13 (Case 7).—Showing, under high magnification, active gliosis taking place about embryologically defective structure.

Bellevue Hospital, where eventually the child came under the observation of Dr. John Hartwell, who later performed an operation for the relief of the hydrocephalus.

In the family history it is perhaps significant that the Wassermann reaction in the blood of the mother was found to be positive, but this examination was made only after operation had been done.

The mother was in ordinary good health at this time and the father died a short time before of spinal meningitis. No definite venereal history was obtained. As a predisposing cause of the hydrocephalus the traumatic influence

of an instrumental delivery may possibly be considered. The patient was the only child and the mother gave no history of abortion or miscarriage.

Physical Examination.—The appearance of the patient at the time of admission to the hospital was that of a hydrocephalic, female infant, well nourished, but very weak and listless. There was an abundant growth of hair.

The head in its greatest circumference measured 52 cm. The fontanels were open; the anterior, bulging and fluctuating, measured 16.5 cm. in its transverse diameter; the posterior was 3 cm. across.

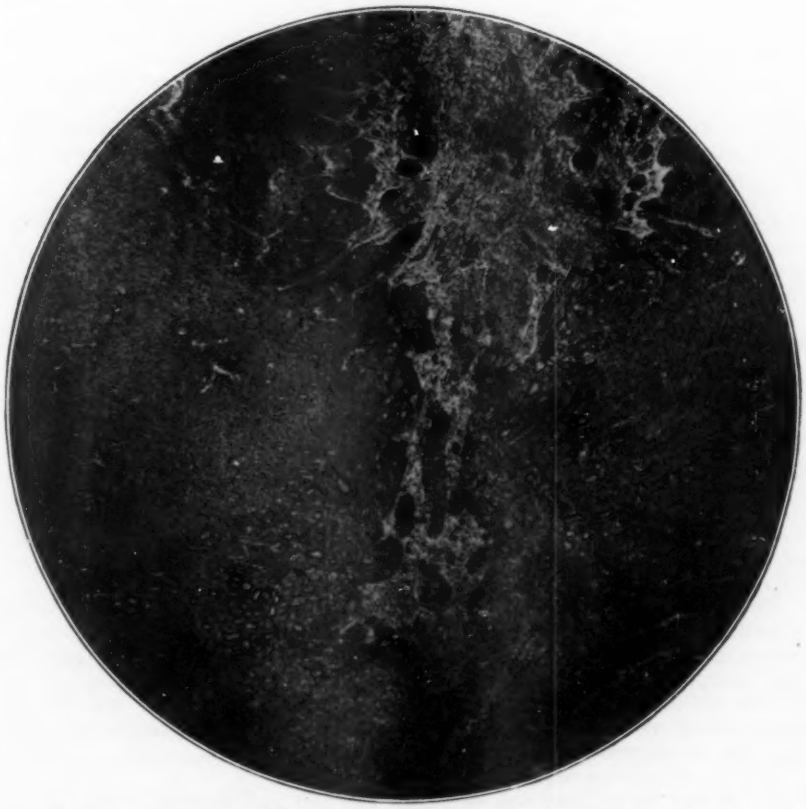

Fig. 14 (Case 8).—Section of medulla in the midolivary region of the fourth ventricle, showing complete obliteration by a central gliosis.

The eyes showed external strabismus, the defect being more marked in the right than in the left eye. The conjunctivae were blue and congested. Examination of the eyegrounds showed the absence of choked disk. The vessels were slightly congested but there were no hemorrhages or papilledema.

The nose, ears and tongue were normal. There was no rigidity of the muscles of the neck; the hands were flexed and thumbs adducted. There was no abnormality of the lower extremities except webbing between the second and third toes of the left foot. The knee jerks were somewhat exaggerated.

The heart, which showed poor muscle tone, was otherwise negative. The action was slow, regular, and there were no murmurs. The lungs were clear.

October 10 a lumbar puncture was made and 10 c.c. of a blood-tinged fluid withdrawn. The records do not show that a Wassermann test was made.

Operation.—October 22, Dr. John A. Hartwell performed an operation designed to drain the ventricles into the jugular vein. The proceeding was as follows:

A section of a vein 4 inches in length was taken from the mother's arm, and this was sutured, one end to the cardiac end of the external jugular vein of the patient, the other end carried by means of a cannula and sutured to the dura mater, the cannula itself being made to enter the lateral ventricle and the external jugular vein, which effectually drained the cerebral cavities.

A considerable amount of cerebrospinal fluid was lost during the operation, so that the fontanels, which previously had been hard and bulging, were, after the procedure, soft and flaccid, while the diameter of the head was reduced to 51 cm.

The fluid in the ventricles was confined under pressure, but the subarachnoid space appeared to be empty. The partial collapse of the skull was due in a measure to loss of cerebrospinal fluid during the operation, but this condition continued afterward, thus demonstrating the fact that drainage had been established and maintained.

The child's condition, however, was not more satisfactory than before the operation, and the weakness and depression, which had marked the case from the beginning, continued until death ensued the following day. The temperature of the patient continued practically normal during the whole period of observation.

Necropsy.—A full necropsy was refused by relatives, but the report as made by Dr. G. H. Wallace is as follows:

	Cm.
Greatest circumference of the head	51
From glabella to external occipital protuberance	33
Distance between mastoid processes	34
Anterior fontanel (depressed)	9 by 14
Posterior fontanel	1.5 by 1

A linear incision 4 inches in length extended downward parallel to the external carotid artery from a point 2 cm. above and 1 cm. posterior to the tip of the right mastoid, which was sutured and clean.

On opening the incision the right external jugular vein was found to be sutured to a transplanted vein which was attached to a cannula at its upper extremity, the cannula itself extending through the brain substance into the right lateral ventricle. The tissues of the scalp were otherwise normal. On opening the skull the dura was found to be adherent to the bone, the cerebral cortex was flattened and fissures and sulci were practically obliterated. In places the cortex was so thin as to be almost transparent.

The cranial nerves were softened and edematous.

The bridge of the nose was depressed and alae nasi flattened. The hair of the head was fine and abundant and extended over the forehead on each side to the supra-orbital ridge.

Macroscopically, the pathologic specimen revealed obliteration of the fourth ventricle by a tumor formation, and complete occlusion of the lower portion of the aqueduct of Sylvius, which showed marked dilatation in front of the posterior corpora quadrigemina. The dilated aqueduct opened into a greatly distended third ventricle, and the wall of the lateral ventricles was stretched to extreme thinness.

Structurally the lesion causing occlusion in this instance is practically identical with that of Dr. Hartwell's previous case, and the same invasion of surrounding tissues by choroidal cells is to be observed microscopically, but the cerebellum does not participate to the same extent as in Case 4.

The fourth ventricle and lower part of the aqueduct were involved in the obliterating process, and while the upper part of the aqueduct was open, active gliosis, with cell proliferation and fibril formation, was a marked feature.

The choroid plexuses did not show any demonstrable changes.

SUMMARY

Reviewing the facts as they appear from our own observations and from a survey of the literature, it seems not unreasonable to attribute many, if not the majority, of cases of the congenital form of this disease to closure of the aqueduct of Sylvius through proliferation of the glia and ependymal tissues, or to invasion of the fourth ventricle by tumors having their origin in the floor, the choroid plexus or the membranous roof of the ventricle.

Those cases, also, developing acutely in previously healthy adults and older children, and which have shown microscopically the same pathologic changes, may have been brought about in the same manner, namely, through the stimulation by some chemical poisoning of a tissue which is embryologically defective. These cases, no doubt, differ etiologically from that large group following meningitis, the acute infectious diseases and those due to bacterial invasion of the brain substance and ependyma from extension of suppurative processes of the middle ear or cells of the mastoid, frontal and ethmoid bones.

A consideration of the life processes of the cell offers an explanation of the occurrence of many cases of congenital and acquired closure of the sylvian aqueduct.

Metabolism is nowhere more delicately expressed than in the highly complex chemical reactions of the cells of the central nervous system; and while knowledge of these reactions is still far from complete, it is, nevertheless, conceivable that any slight noxious influence may be sufficient seriously to disturb the latent forces of the glia cells, resulting in the dominating influence of one or another of the processes residing in these cells. These life processes may be divided into the nutritive, the formative and the functional activities. In the first, which involves an appropriation of nutritive substances from the blood, potential energy is stored up and is subsequently translated into formative or functional activity, as represented by cell division, on the one hand, or by functionation of the specialized cell, on the other. In cells which have become highly specialized, as the nerve cell, gland cell and muscle cell, the potential energy of the cell body is converted into the predominating activity, and the formative process is held in abeyance, and, so long as the normal relation between synthesis and catalysis is maintained, functional activity of the cell remains in a state of constancy. On the other hand, in those cells not highly specialized, as the ependymal, glia and the connective tissue cells, formative activity is easily awakened, and so it happens that in many cases of hydrocephalus, stimulation of these cells by some irritating substance results in an active proliferative process which involves not only the ependyma but the subependymal tissues of the aqueduct of Sylvius, as seen in the obliterative glioses which figure conspicuously in the accompanying photographs.

The exact nature, source and means of access of such damaging stimuli are not always readily determined, and, barring the possible influence of syphilis and bacterial invasion of the meninges, ependyma and choroid plexuses, there remain to be studied more closely the causal effects of circulating toxic byproducts of fetal or maternal metabolism, and variation in function of the endocrine glands.

Syphilis has not operated as a known etiologic factor in this sequence, with the exception of Case 6, in which *Spirochaeta pallida* have been found in the cerebral tissue. The Levaditi method has failed to show the presence of spirochetes in two other cases (2 and 7) which it was thought might be explained on the ground of a luetic infection. The clinical records do not include reports of the Wassermann reaction in the blood or spinal fluids of the parents of these children.

A CONTRIBUTION TO THE DIAGNOSIS OF SPINAL CORD TUMORS IN CHILDHOOD*

RACHEL L. ASH, M.D.
SAN FRANCISCO

During the last fifty years, as a result of continuous study of the physiology and pathology of the spinal cord, the clinical diagnosis of intravertebral tumor has been made with increasing frequency. Many case reports are now available. From these it would appear that growths within the spine occur exceptionally in childhood, differing in this respect from those within the cranium. Stursberg[1] collected from the literature reports of 141 operations; one patient was under 10 years, and only six were between the ages of 10 and 15. Schultze,[2] in his series, introduced but one case, a girl of 4 years, while the age of the youngest in Nonne's[3] latest report of sixteen operations was 19 years. The occurrence of these tumors in early life was not noted in Flatau's[4] recent monograph.

Postmortem statistics differ from these very occasional findings in the clinic. Among 251 collected reports of tumors of the spinal cord, Schlesinger[5] found 33 (13 per cent.) in patients between the ages of 1 and 9 years, and 27 (11 per cent.) between the ages of 10 and 15. The earlier compilations of Gowers and Horsley[6] (10 per cent.) and Lloyd and Mills[7] (12 per cent.) show practically the same ratio.

The following case report is a contribution to the study and diagnosis of spinal cord tumors in childhood.

REPORT OF CASE

Clinical History.—A. L. (No. 8241, University of California Hospital), aged 1 year, 11 months, birthplace, San Francisco, was admitted Nov. 13, 1914; died Nov. 21, 1914. Under observation eight days.

Family History.—Negative; no tuberculosis, nervous disease or rheumatism; four other children, living and well.

---

* Submitted for publication March 30, 1917.

* From the Department of Pediatrics, University of California Medical School.

1. Stursberg: Centralbl. f. d. Grenzgeb. d. Med. u. Chir, 1908, **11,** 91.
2. Schultze, F.: Mitt. a. d. Grenzgeb. d. Med. u. Chir., 1903, **12,** 153.
3. Nonne: Deutsche. Ztschr. f. Nervenh., 1913, **47,** 436.
4. Flatau: Wirbel-und Rückenmarksgeschwülste, in Lewandowsky's Handbuch der Neurologie, Ed. 1, Berlin, S. Karger, 1912, **2.**
5. Schlesinger: Beitrag zur Klinik der Rückenmarks-und Wirbeltumoren, Vienna, 1898.
6. Gowers and Horsley: Med.-Chir. Tr., London, 1888.
7. Lloyd and Mills: Spinal Tumors, in Pepper's System of Practical Medicine, Ed. 1, Philadelphia, Lea, 1885-1886.

Past History.—Birthweight, 12 pounds; breast fed more than six months; began to walk and talk at the usual time. The first illness, measles, some eight months prior to admission, was not severe; there were no immediate after-effects.

Present Illness.—For four months the patient had been continuously ill. His sickness began when "he caught cold which settled in the glands of his neck and caused his head to lean a little to the right. Sometimes he had pain in the right shoulder." After fourteen days, the right hand was noticeably weak. According to a physician who saw him shortly afterward, it was practically useless. The patient had beside, at this time, a sore throat, much enlarged cervical glands, and a temperature of 100 F. After six weeks, weakness, which had since increased progressively, was noted in the right leg. Three months after the beginning of his illness the patient was confined to his bed. About this time the child cried a great deal, for about one week, with pain or itching in the right hand and left shoulder, which was relieved by rubbing. Soon his left hand became weak. Since one month, there had been intermittent fever with night sweats. Urination had been painful; there was retention at times; the bowels had been normal; the appetite, very good until recently. There had never been any cough, convulsions, vomiting or night cries. There had never been any severe pain. The mind had always been clear.

Physical Examination.—(Nov. 14 to 19, 1914). A well-developed, well-nourished baby, with a somewhat worried expression, mentally alert, lay perfectly quiet with his head inclined to the right. When disturbed, he rolled his head from side to side, crying "No! No!" in a good clear voice. He put out his tongue when asked. The neck, held stiff, was rigid, and although he could turn his head, he never raised it up. There was no rigidity of the lower spine. Pressure over the lower cervical vertebrae elicited pain, but this was not found elsewhere along the vertebrae. There was no kyphosis or scoliosis. Many discrete, enlarged cervical glands were apparent on both sides of the neck, but beneath the angle of the right jaw and under the right sternocleidomastoid there was a mass of glands that were noticeably enlarged and tender. Glands were also palpable in the axilla and inguinal regions. Both sides of the chest moved equally. Heart, lungs, and abdomen were negative. The temperature ranged from 38.2 to 39.8 C.; pulse 120 to 140; respirations 22 to 35. On repeated examination the urine was always negative. The leukocytes varied from 10,000 to 20,000, and the neutrophils from 44 per cent. to 88 per cent. The Wassermann test was negative in the blood serum. The von Pirquet test was strongly positive to both human and bovine tuberculin. A roentgenogram of the cervical spine showed no changes in the vertebrae.

Examination of the Nervous System.—The child lay as above described, and with his eyes usually turned to the right. The upper extremities were extended, the lower flexed at the knees. Percussion of the head showed nothing unusual; there were no painful points. The right eyeslit was narrower than the left, but there was no strabismus or nystagmus. The pupils reacted promptly to light and accommodation. The corneal reflexes were lively. Fundus examination revealed a slight blurring of the edges of the disks and a mild venous hyperemia — low grade optic neuritis. The right half of the face drooped more. Hearing was normal. The tongue was extended in the median line. The palatal and pharyngeal reflexes were present.

No voluntary movements were initiated. If the right arm was raised, the patient would slowly lower it; no motion seemed possible in the opposite limb. If either leg was extended, he would gradually flex it. There was flaccid paralysis of both upper extremities. The right wrist was flexed, the right hand was a typical "ape hand." The left hand and fingers were permanently flexed. Spasm was present in the adductors of the thigh, at the knee, and in both ankles. Kernig and Brudzinski signs were not present. Ankle clonus was present, more marked on the right. The tissues were generally

flabby throughout, but there were no atrophies, apparently, except in the right interossei, thenar and hypothenar groups. (Electrical reactions were not determined.)

The patient's responses to heat and cold and to touch were unreliable. To pin prick, without doubt, there was greatly diminished sensation from the second intercostal space and the second dorsal vertebra to the soles of the feet. Only on the dorsal surface of the fingers and on the anterior surface of the thighs, did any sensation appear to be present.

Tendon reflexes were everywhere present, livelier on the right than on the left.

Neither the abdominal nor cremasteric reflex was present, and only occasionally could the plantar be elicited. Positive Babinski and Oppenheim signs were found.

Alternate flushings on the right and on the left cheek, and profuse sweating over the face, head and upper part of the body were noted.

In all, four spinal punctures were made, the last being a dry puncture. The fluid had always the same characteristics. It was obtained under low pressure, and was yellow and clear on withdrawal. The immediate cyto count averaged 250 cells per c.mm., 15 per cent. polymorphonuclears, and 85 per cent. mononuclears. If heated, the fluid became solid at once. If left to stand, clots formed quickly and the fluid solidified in a short time. No tubercle bacilli or other organisms were found.

Nov. 20, 1914, the temperature suddenly rose to 41.6 C., pulse 152, and respirations 40 to 50. A maculo-papular eruption appeared on the dorsal surface of the hands and forearms. Toward night the child became semiconscious, the right pupil was larger than the left, but both still reacted to light. Strabismus and rolling of the eyes occurred intermittently. There was twitching of the body, and especially of the right face. Cheyne-Stokes breathing was observed. Nov. 21, 1914, death occurred.

CLINICAL DIAGNOSIS

The conditions presented in the foregoing case are readily separable into (1) general signs, and (2) signs specially referable to the nervous system.

1. *General Signs.*—The irregular fever, the enlarged cervical lymph nodes, the positive von Pirquet, together with the history of an illness in a previously healthy child in the second year of life, which appeared four months after measles — these signs all point, in the absence of a positive Wassermann reaction and other evidences of congenital syphilis, to a *tuberculous infection.*

2. *Signs Referable to the Nervous System.*—These may be divided into (*a*) those of spinal origin, and (*b*) those of cerebral origin.

(*a*). Spinal Signs: A pain, or paresthesia, in the right shoulder preceded a unilateral paralysis, which began in the right upper extremity, and passed to the right lower extremity. Then followed a weakness of the left shoulder girdle, and finally a complete paraplegia. Spasticity was demonstrable in the lower extremities. Sensibility to pain seemed almost completely absent below the second intercostal space (C_5). Tenderness was sharply localized over the lower cervical vertebrae. "Coagulation massive et xanthochromie" of the spinal fluid appeared as a constant phenomenon.

The Brown-Séquard syndrome, the secondary paraplegia, the segmentary distribution of the disturbances in sensation, and the localized vertebral tenderness, denote a *compression of the spinal cord, due to a circumscribed lesion.* (Schultze,[2] Oppenheim,[8] Bruns.[9]) The heavy albuminous deposit and the yellow color in the spinal fluid are also significant of pressure (Nonne[3]).

Disease external to the vertebrae, in the vertebrae themselves, or within the vertebral canal, causes compression of the spinal cord. No tumor growths of extraspinal origin were observed which by proximity or metastasis could infiltrate the bony structures. Caries and osteomas and other tumors of the vertebrae are eliminated by the short duration of the illness, the absence of severe pain, of kyphos and scoliosis, and the negative findings in the roentgenogram.

A condition within the spinal canal must then have produced the compression, and to fit the clinical picture here presented, must have involved the cord or the meninges, or both. To be considered, therefore, are meningomyelitis, pachymeningitis cervicalis hypertrophica, and tumor. An acute meningomyelitis, due to a specific organism, manifests itself as a primary disorder with an acute onset, or as a complication of an already existing process; it presents bilateral symptoms early, and, especially in children, runs a much shorter course. A myelitis luetica, limited to the spinal cord and its meninges, does not occur in very early life, and here there were no congenital stigmata and the Wassermann was negative. Tuberculous myelitis is a disseminated lesion. Pachymeningitis cervalis hypertrophica is bilateral from the start and is marked by its extreme chronicity. By the exclusion of meningomyelitis and pachymeningitis the diagnosis of *spinal tumor* is warranted.

Was the tumor intramedullary or extramedullary? The absence of definite root pains and the rapid extension of the cord symptoms suggest an intramedullary lesion; but the long-continued radicular palsy in the right upper extremity, the early right unilateral signs, the localized tenderness in the cervical vertebrae, and the tendency of the process to spread transversely and not upward (Schultze[2]), may as readily be attributed to an extramedullary lesion.

Location of the tumor: The *main-en-griffe* denotes at once the seventh and eighth cervical and first dorsal segments, with the corresponding roots of the brachial plexus. The upper limit of the segmentary distribution of the sensory disturbances (C_5) confirms this. Further evidence for this situation is the involvement of the cervical

8. Oppenheim: Lehrbuch der Nervenkrankheiten, Ed. 5, Berlin, S. Karger, 1908.

9. Bruns: Geschwülste des Nervensystems, Ed. 2, Berlin, 1908.

sympathetic — narrow right eyeslit, alternate flushings of both cheeks, and profuse sweating in the upper half of the body.

(*b*). Cerebral Signs: The paresis of the right side of the face, the optic neuritis of low grade, the late strabismus, the twitching and irritation of the right pupil, and the high lymphocyte count in the spinal fluid indicate meningeal irritation. A tuberculous infection was undoubtedly present elsewhere in the body, and may have been metastatic here in the brain and spinal cord as an early meningitis. The long period of unclouded consciousness, the absence of convulsions, headache and vomiting make this meningitis a matter of minor importance.

Clinical Diagnosis. Intramedullary or Extramedullary Tumor of the Spinal Cord. Compression Myelitis. Meningitis, Probably of Tuberculous Origin.

Abstract of Necropsy Report.—(Dr. E. H. Falconer).

Right Lung: In the middle lobe, and occupying about one-half of the lobe, is a hard, noncrepitant area, 4 by 2 cm. On section this tissue is necrotic and a purulent material exudes from its center. A definite capsule separates it from the surrounding tissue.

Left Lung: Scattered throughout the lung tissue are small, round areas 0.5 to 2.0 mm. in diameter, some caseous, others calcareous.

Peribronchial Lymph Nodes: These are much enlarged and hard, but show no sign of breaking down.

Intestines: The ileum shows several round, irregular, ulcerated areas 6 by 7 mm. in diameter. They seem to correspond in distribution to Peyer's patches. In the wall of the ulcer are many small, nodular, caseous areas.

Mesenteric Lymph Nodes: These are markedly enlarged and everywhere matted together. On section they show caseous areas, but with no evidence of breaking down.

Brain: There are slight congestion over the cortex and marked edema at the base, and a few scattered tubercles with considerable exudate in the meninges of pons and the circle of Willis. Immediately superior to the posterior portion of the corpus callosum, in the cingulum, and situated for the most part in the gray matter of the convolution, is a small, round, grayish-yellow mass of tissue, about 1.5 by 2.0 cm.

Microscopic Examination.—Sections of the cerebral tissue between gyri show a pia distended by exudate. This exudate consists principally of lymphoid cells and is well marked about the vessels. Large, irregular masses of caseous necrosis are scattered in the exudate. These are localized but not typical tubercles.

The Tumor: There is a zone of inflammatory reaction about the mass, as evidenced by epithelioid cells, a few lymphocytes, and congestion of the vessels. At the periphery of the tumor mass, there is an artery whose lumen is nearly closed by an obliterating endarteritis. The tumor itself consists of a pale-staining, necrotic tissue with occasional giant cells.

Cord: In the cervical enlargement, at the level of the third, fourth and fifth cervical vertebrae, considerable bulging shows beneath the dura mater, which is much congested in this area. On slitting the dura, the white substance is so soft that it exudes through the cut surface. At this level is seen an olive-shaped tumor mass, 2.5 by 1.2 cm., occupying the greater portion of the canal and flattening the cord posteriorly. It is extramedullary and does not infiltrate the cord substance proper. It has not infiltrated or become attached to the nerve roots. On section it is grayish-white, homogeneous, and of caseous appearance; there is no evidence of liquefaction.

Microscopic section of the tumor shows it to consist of a central caseous material, surrounded by epithelioid and giant cells and some lymphocytes. Beyond this there is a zone of fibrous tissue. The spinal meninges are heavily strewn with typical lymphoid tubercles, especially in the cervical and dorsal regions.

Pathologic Diagnosis.—General miliary tuberculosis. Caseous and miliary tuberculosis of lungs. Tuberculosis of bronchial and peribronchial lymph nodes. Miliary tuberculosis of spleen, liver and kidney. Tuberculous ulcers of the intestines (ileum). Miliary tubercu-

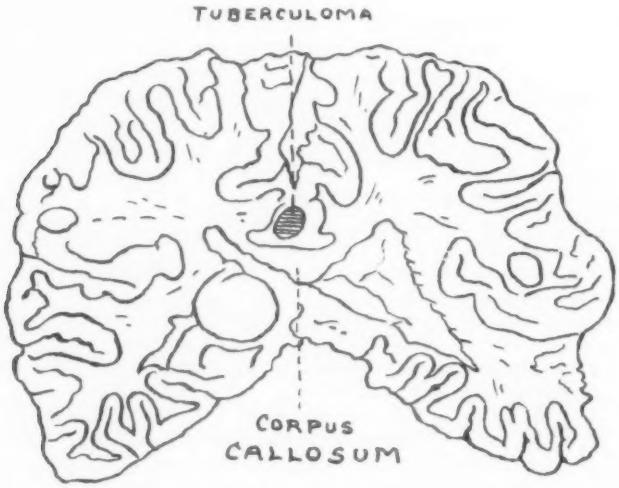

Fig. 1.—Tuberculoma of cerebral cortex (author's case).

losis of the appendix. Tuberculosis of the mesenteric lymph nodes. Tuberculosis of the meninges of the brain and spinal cord. *Tuberculoma of the cerebrum* (cingulum), *tuberculoma of the spinal cord.*

DISCUSSION

Several rather uncommon features of this case need special consideration.

The Absence of Pain.—Pain, as a symptom, was formerly considered necessary for the establishment of a diagnosis of spinal tumor. Within the last few years, however, numerous cases have been reported in which no pain has been present. (Nonne,[10] Flatau,[4] Panski,[11] Newmark,[12] Collins and Marks,[13] and others.) "We must realize that pain and other classical data are valuable but not essential phenomena, but that the essential elements (first emphasized by Schultze) is the deter-

10. Nonne: Neurol. Centralbl., 1908, **27,** 749.
11. Panski: Neurol. Centralbl., 1912, **31,** 1208.
12. Newmark: Berl. klin. Wchnschr., 1914, No. 43, p. 739.
13. Collins and Marks: Am. Jour. Med. Sc., June, 1915, p. 613.

mination of a gradually progressive motor and sensory paralysis, the upper pole of which, despite increase of cross section and intensity, varies slightly if at all." (Collins and Marks.[13])

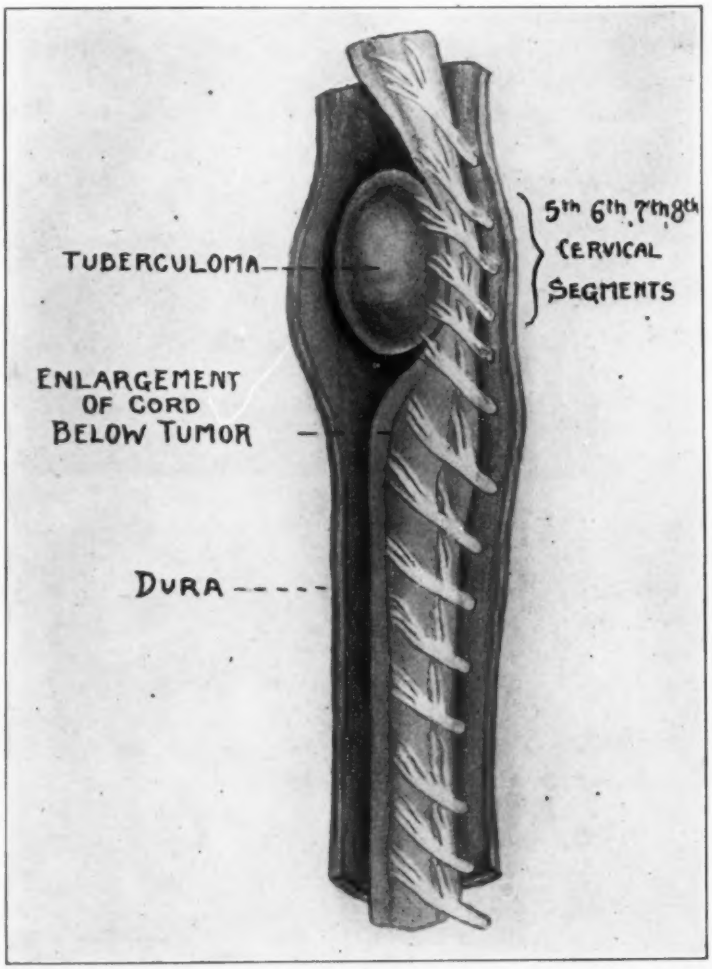

Fig. 2.—Extramedullary tuberculoma of spinal cord (author's case).

Xanthochromia of the Cerebrospinal Fluid with Massive Coagulation.—Of late much attention has been paid to this appearance, first described by Froin[14] in 1903. (Mestrezat,[15] Nonne,[3] Mix,[16] Bromer,[17]

14. Froin: Gaz. d. hôp., 1903, **76,** 1005.
15. Mestrezat: Le liquide cephalo-rachidien, normal et pathologique, Paris, 1912.
16. Mix: Clinics of John B. Murphy, April, 1915.
17. Bromer: Am. Jour. Med. Sc., March, 1916, p. 378.

Ayer and Viets.[18]) Spinal fluid of a yellow color may be divided into those with great amounts of globulin which coagulate spontaneously, and those which do not coagulate, according to Sprunt and Walker,[19] who recently studied 100 cases of xanthochromia. Fluids of the former type present what is known as Froin's Syndrome, and also as Nonne's Compression Syndrome. It always denotes a compression of the spinal cord above the site of puncture, with a consequent stasis of the fluid below the compression. The fourth lumbar puncture in this case was "dry" — there was no more fluid to evacuate. An increased lymphocytosis as an accompanying condition signifies an inflammation of the meninges. Here it was diagnostic of a tuberculous meningitis.

Position and Nature of Spinal Cord Tumors in Childhood.—Schlesinger[5] finds that meningeal tumors are more frequent in adults than in children, in whom the extramedullary tumors are usually extradural lipomas or masses of tubercle. Up to the fifth year, tumors within the cord are generally tuberculomas; later, sarcomas predominate. The tumor here under consideration was unusual both in position and pathology.

The Cerebral Growth.—This occupied a silent area, the cingulum of the cortex. No symptoms were here noted that could be traced to a brain tumor. Ziehen[20] is authority for the statement that more than 50 per cent. of all brain tumors in children are solitary tubercles, that they are multiple, that they rarely invade the cortex, but when they do, usually elect the paracentral lobule.

Prognosis.—The prognosis in spinal cord tumors of tuberculous origin is grave. Cases of temporary cure by operative procedure have been reported. (Starr,[21] Bonniger and Adler,[22] Veraguth and Brun.[23]) Until our knowledge of tuberculosis, especially tuberculosis of the central nervous system, becomes more complete, this condition will remain hopeless.

CONCLUSION

Of special clinical interest in this case report is the fact that a tumor of the spinal meninges, which was diagnosed during life, was the first manifestation of a generalized tuberculosis in a child of 19 months. Xanthochromia with massive spontaneous coagulation and pleocytosis was an attending syndrome.

240 Stockton Street.

18. Ayer and Viets: Jour. Am. Med. Assn., Dec. 6, 1916, p. 1707.
19. Sprunt and Walker: Bull. Johns Hopkins Hosp., February, 1917, p. 80.
20. Ziehen: Handbuch der Nervenkrankheiten im Kindesalter, Ed. 1, Berlin, S. Karger, 1912.
21. Starr, M. Allan: Am. Jour. Med. Sc., June, 1895, p. 613.
22. Bönniger and Adler: Med. Klin., 1911, Nos. 18-19, p. 279.
23. Veraguth and Brun: Cor.-Bl. f. schweiz. Aerzte, 1916, No. 14; abstr., Jour. Am. Med. Assn., Nov. 26, 1916, p. 1587.

PHYSIOLOGY OF THE NEW-BORN INFANT*

FRITZ B. TALBOT, M.D.
BOSTON

There has been an increasing amount of investigation on the metabolism of the new-born infant during the past few years, but only recently enough evidence has accumulated to be of any practical value to the general practitioner. This communication deals only with that part of the metabolism which is known as the respiratory metabolism, and from which the energy metabolism may be calculated.

The earliest study of the respiratory metabolism of the new-born infant was in 1877 by Forster,[1] who measured the carbon dioxid output of an infant 14 days old. Since then many investigators have recorded observations made by them, using different forms of apparatus. The most important of these was published in Danish in such an inaccessible form that it was overlooked in recent writings on the subject. This work by Hasselbach was reported in 1904. The original work, which is given in full in Publication 233 of the Carnegie Institution of Washington, was translated into English under the direction of Dr. Francis G. Benedict and corrected by Hasselbach himself. A detailed report of the work on the new-born done by Benedict and the writer, and a complete discussion of the literature on the physiology of the new-born may also be found in this publication.

It is necessary, before treating the subject matter of this paper, to define some of the technical terms used later.

Basal metabolism means the energy production of an infant in complete muscular repose, and may be obtained only when the baby is asleep. In one instance we found that during crying the metabolism was three times as great as when the baby was sleeping quietly. An ideal state in which to determine the basal metabolism would be the so-called "post-absorptive state" of adults; that is, when the stomach is empty; but this is rarely possible with infants, as they usually sleep only when the stomach is full. It is obvious, therefore, that only figures of the minimum or basal metabolism can be used for comparison in any instance in which the metabolism is determined for less than the so-called twenty-four-hour observation.

The *respiratory quotient* means the volume of carbon dioxid expired, divided by the volume of oxygen used. It was found when

* Submitted for publication April 9, 1917.
* From the Nutrition Laboratory of the Carnegie Institution of Washington.

1. Forster: Amtlicher Bericht der 50 Versammlung deutsche Naturforscher u. Aerzte in Munchen, Munich, 1877, p. 355.

pure carbohydrate was burned up outside the body that the volume of oxygen necessary for its combustion was always the same as the volume of carbon dioxid given off as a result of the combustion. The respiratory quotien of carbohydrate $\frac{CO_2}{O_2}$ is, therefore, always 1.00. The respiratory quotient of fat is 0.713, and of protein 0.801. Tables have been made from which the amount of the different food components that are being burned up in the body may be estimated, for each respiratory quotient. For example, when a respiratory quotient is 0.90, two-thirds of the energy used up comes from carbohydrate and one-third from fat.

Colostrum plays such an important part in the first three days of life that it is necessary to give a few essential facts concerning it, based on a study of the literature. The mother's breasts at birth contain only a small quantity of colostrum, which is increased under the stimulation of sucking, until the breast milk "comes in," on about the third or fourth day after birth. The energy or heat value of colostrum is about 650 calories per liter, although it may be considerably higher at times.[2] The amount of colostrum during the first twenty-four hours after birth is between 4 and 6 c.c., and gives approximately 3¼ calories. During the second day the baby receives from 78 to 129 c.c. of colostrum, or 51 to 84 calories. On the third day the amount of colostrum varies between 199 and 238 c.c., which give 129 to 154 calories. Colostrum, therefore, gives the new-born infant only a minimal amount of energy, insufficient to supply its energy requirements. It is quite natural, therefore, that it should lose weight during the colostrum period.

Loss of weight: Nearly all infants lose weight during the first few days of life, some losing more than others. This loss of weight is of two kinds: (1) "mechanical" and (2) "physiologic." The mechanical loss represents material which may be collected and weighed, and is due principally to the passage of urine and meconium, and at times to the vomiting of allantoic fluid, swallowed by the infant before birth. There is also a loss due to the evaporation of water from the skin. The physiologic loss of weight is that due to the actual oxidation of body substance as a result of metabolism, and to the fact that an infant is virtually starved during the colostrum period. The colostrum which an infant receives does not give enough food to supply the necessary energy to keep it warm. The energy and body heat must, therefore, be supplied in no small part by the glycogen in the liver and tissues. This glycogen is quickly used up and the energy is then obtained from the body fat. (See respiratory quotients, in Table 1, of the first, second and third days of life.) The body fat now assumes a very important rôle in the economy of the new-born infant, as there is not enough

2. Langstein: Rott and Edelstein: Ztschr. f. Kinderh., 1913, **7**, 210.

sugar and glycogen in the body to supply the energy necessary to keep a baby warm for more than a few hours. This fat, which was deposited during the last month of intra-uterine life, is, therefore, the storehouse of fuel and must be present in large enough amounts to supply energy until the breast milk "comes in" to take its place. Clinical experience bears out the inference that infants without a good layer of fat at birth should receive food at the earliest possible moment. This is especially true of prematurely born infants who are almost entirely devoid of fat and should be fed at a very early hour in order to conserve their strength. After the establishment of the milk supply there is a gain in body weight, the respiratory quotient rises, and the energy used by the infant is now obtained from the mother's milk instead of from its body tissues. Table 1 gives the average respiratory quotient of 105 babies.

TABLE 1.—AVERAGE RESPIRATORY QUOTIENTS OF INFANTS DURING THE FIRST EIGHT DAYS AFTER BIRTH

	First Day	Second Day	Third Day*	Fourth Day	Fifth Day	Sixth Day	Seventh Day	Eighth Day
Body weight, Kg.	3.48	3.41	3.32	3.34	3.43	3.51	3.54	3.82
Respiratory quotient	0.80	0.74	0.73	0.75	0.79	0.82	0.81	0.80

* Breast milk commences to "come in."

Energy requirements: It has long been thought that the "energy requirements of infants relative to the weight, are greatest during the first months of life, but gradually diminish, until at the end of the first year they are only three-fourths as great as during the first month.[3] Although this may be relatively correct after the third month, quite the reverse is true of the first and second months of life. A new-born infant requires approximately 62 calories per kilogram of body weight per twenty-four hours, but needs more as he grows older. We found that the *basal* energy requirement of infants from 1½ to 6 days of age was 44 calories per kilogram of body weight, whereas the average of a 4 to 5 months-old infant is in the neighborhood of 55 calories. It is quite probable that this discrepancy is due to the fact that the new-born infant grows fat on the very abundant supply of breast milk received during the early months of life, but muscles begin to replace the fat as the infant grows and represent a larger proportion of the body weight of the infant than at birth. The amount of heat produced depends on the amount of muscle and other "active protoplasmic tissue" in the body, and not on the fat, the heat per unit of body

3. Holt and Howland: Diseases of Infancy and Childhood, New York and London, 1916.

weight being in inverse proportion to the amount of body fat. The fatter the baby, therefore, the fewer the calories per kilogram of body weight will it require.[4] The basal metabolism of the new-born infant is also much lower than that of older infants when it is calculated per square meter of body surface, or as total heat per twenty-four hours.

A great deal of time was spent in studying the figures obtained from observations on the new-born to find, if possible, some law that might be applicable to them. These show that the shorter infants produced less heat per square meter of body surface than the longer

TABLE 2.—BODY-SURFACE COMPUTED FROM THE BODY-WEIGHT BY THE LISSAUER FORMULA ($10.3\sqrt[3]{wt.^2}$)

Body Weight, Kg.	Body Surface, Sq. M.	Body Weight, Kg.	Body Surface, Sq. M.	Body Weight, Kg.	Body Surface, Sq. M.	Body Weight, Kg.	Body Surface, Sq. M.
2.00	0.163	2.80	0.205	3.55	0.239	4.30	0.272
2.05	0.166	2.85	0.207	3.60	0.241	4.35	0.274
2.10	0.169	2.90	0.210	3.65	0.244	4.40	0.277
2.15	0.172	2.95	0.212	3.70	0.246	4.45	0.279
2.20	0.174	3.00	0.214	3.75	0.249	4.50	0.281
2.25	0.177	3.05	0.217	3.80	0.251	4.55	0.283
2.30	0.179	3.10	0.219	3.85	0.253	4.60	0.285
2.35	0.182	3.15	0.222	3.90	0.255	4.65	0.287
2.40	0.184	3.20	0.224	3.95	0.257	4.70	0.289
2.45	0.187	3.25	0.226	4.00	0.260	4.75	0.291
2.50	0.190	3.30	0.228	4.05	0.262	4.80	0.293
2.55	0.192	3.35	0.231	4.10	0.264	4.85	0.295
2.60	0.195	3.40	0.233	4.15	0.266	4.90	0.297
2.65	0.197	3.45	0.235	4.20	0.268	4.95	0.299
2.70	0.200	3.50	0.237	4.25	0.270	5.00	0.301
2.75	0.202						

infants, after the first day and a half of life. The heat production per square meter of body surface was, therefore, divided by the length of the infant, and the average of the resultant figure was 12.65 calories. This figure was, therefore, taken as a constant and the following formula devised:

Total calories = length $\times$ 12.65 $\times$ body surface.

The body surface may be calculated from the weight of an infant by using Lissauer's formula, given in Table 2, in which the body surface is worked out for the various weights of infants. The basal

4. Benedict and Talbot: Publication 201, 1914, Carnegie Institution of Washington, and Am. Jour. Dis. Child., 1914, **8**, 1.

metabolism may be calculated for any new-born infant of known weight and length by the use of the formula, length × 12.65 × body surface, and has been found to come within 6 per cent. of the basal metabolism actually measured in the respiratory chamber.

The metabolism during the first twenty-four to thirty-six hours of life was, on the whole, much lower than later, even when it was obtained with the same infant on successive days. In some instances this could be attributed to the fatigue incident to hard labor, but in the majority of instances the low metabolism during this period could be traced to exposure and a consequent subnormal temperature. A series of rectal temperatures showed that heat was lost from the body shortly after the water bath, when the body temperature began to fall. A subnormal temperature did not result in the more vigorous infants, who either commenced to cry or to shiver when they were cold, and thus kept the body temperature normal and produced enough heat to offset the loss from the skin. In less vigorous infants, on the other hand, the temperature became subnormal and their metabolism was correspondingly diminished, so that coincidently there must have been a depression of all the body functions, which was detrimental to the infant. In one instance the temperature dropped to 95.4 F. (35.2 C.). The writer believes, therefore, that the chilling from a water bath is the cause of death of many new-born infants, particularly premature infants, and should be entirely discarded. The baby may be satisfactorily bathed with warm oil, without evaporation and excessive loss of heat from the body.

If the new-born infant does not receive enough food during the first three or more days of life, what should be done? It is obvious that the "mechanical" loss of weight cannot be prevented, but it is quite different with the "physiologic" loss. Of the many methods suggested to prevent this loss of weight, the writer feels that the giving of breast milk within a few hours of birth is the most satisfactory. Baily and Murlin[5] suggest a mixture of cow's milk similar in composition to colostrum, which they have used with favorable results. The writer, however, feels that cow's milk should be avoided, when possible, during the early days of life, since it is a foreign protein, which may be absorbed directly into the blood and start the reaction which will either result in sensitization or immunity. "There is no time in a baby's life in which it is so easy to disturb the digestion or at which it is so difficult to correct the disturbance, if it is once caused."[6] A much better and safer procedure is to use human milk, drawn from a healthy woman.

5. Bailey and Murlin: Am. Jour. Obst., 1915, **71,** 526.
6. Morse and Talbot: The Nutrition and Feeding of Infants, New York, 1915, p. 125.

diluted if necessary with boiled water. Human milk has the advantage of being a homologous protein, of a composition adapted to the needs of the human infant. The writer has used human milk in feeding immediately after birth, for many years, with excellent results. Schick[7] tried the experiment of forcing new-born infants to take breast milk from the spoon and found that he could prevent the usual loss of weight after birth. A third method which may be used, in case human milk cannot be obtained, is to give a sterile solution of sugar and water. Theoretically, dextrose, the sugar present in the blood, is the best one to choose. Lactose and cane sugar are commonly used and have given satisfaction in America and other countries.

SUMMARY

1. The respiratory quotients of new-born infants indicate that the supply of glycogen in the body is quickly used up, and that the energy is obtained in large part from the body fat until the breast milk "comes in."

2. The energy requirements of new-born infants are smaller per unit of body weight than in older infants.

3. The total calories of the basal metabolism of a new-born infant may be calculated from the following formula:

$$\text{Total calories} = \text{length} \times 12.65 \times \text{body surface}.$$

4. Chilling from exposure or a water bath depresses the metabolism and with it all the body functions. A new-born infant should not be bathed in water, and great care should be taken that it is not chilled. Warm oil should be used to clean the body.

5. Since a new-born infant is starved until the breast milk "comes in," weak or premature infants should be fed shortly after birth, preferably with the milk of another woman; but when this is lacking, a 5 per cent. solution of some sugar such as lactose should be given as a temporary expedient.

311 Beacon Street.

7. Schick: Ztschr. f. Kinderh., December, 1915.

THE USE OF THE LONGITUDINAL SINUS FOR DIAGNOSTIC AND THERAPEUTIC MEASURES IN INFANCY *

ALAN BROWN, M.B., AND GEORGE E. SMITH, B.A., M.D.
TORONTO, CANADA

Owing to the fact that the examination of the blood in infants offers considerable aid in diagnosis, and that blood has been shown to be an effective medium for the introduction of therapeutic fluids, it has been necessary to look for other means of entering the blood stream than through the jugular and scalp veins, which are not always accessible, and attempts to enter them quite frequently result in failures. This is especially true in some toxic conditions in which even the external jugular is not to be seen or palpated.

Marfan, in 1898, first employed the longitudinal sinus for the intravenous injection of salt solution. Tobler, in 1915, described in detail the anatomy and technic, and Helmholz of Chicago, in the same year, described a special apparatus, useful in transfusion into the longitudinal sinus.

From the anatomic point of view, the longitudinal sinus offers the most effective route for injection of fluids or obtaining blood. The vein is rigid, cannot be pushed aside, and on entering, one gets a definite sensation of entering a vessel. The tissues that are pierced are the skin and fibrous tissue of the fontanel, each of which are about $\frac{1}{8}$ inch in thickness, so that the average distance into the sinus is about $\frac{4}{16}$ to $\frac{5}{16}$ of an inch. From a series of measurements we found that the sinus grows gradually larger from before backward, so that the anterior part of the fontanel measures $\frac{3}{32}$ inch, posterior part $\frac{3}{32}$ to $\frac{4}{32}$ inch, and 1 inch along the suture posterior to the fontanel, $\frac{5}{32}$ inch, while 2 inches along the suture posterior to the fontanel it measures $\frac{5}{32}$ to $\frac{6}{32}$ inch. It is, therefore, advisable to enter the fontanel at the beginning of the suture just posterior to the fontanel. When one takes into consideration the fact that punctures of the ventricles can be done without harm, the damage that might result from an occasional puncture of the brain substance is negligible.

TECHNIC

The technic employed in entering the sinus is very simple and correspondingly rapid. All that is necessary is a 20 c.c. Luer syringe, with an 18 or 20 gauge needle about $1\frac{1}{4}$ inches long. The needle is

* Submitted for publication April 9, 1917.
* From The Infants' Department and Laboratory, Hospital for Sick Children.

slightly modified in two respects: first, the bevel is made about 45 degrees, so that the point does not enter the sinus deeply. Second, a guard is placed about ¼ inch from the extreme tip of the needle to prevent the latter transfixing the sinus as it passes, sometimes abruptly, through the fibrous layer of the fontanel. In the beginning, to accomplish this, a furrow was filed about the needle at the right distance from the tip, a few turns of fine silk being used to fill the furrow. At the suggestion of Dr. Arnold of Buffalo, an improvement was made on this by using solder to make an elevated ridge around the needle. The latter method is employed at present. We keep on hand a series of three or four needles with the guard varying from ¼ to 5/16 inch from the point.

In entering the sinus the needle is always first attached to the syringe to give precision in handling. If it is expected to use more than one syringe, sterile petrolatum is applied to the tip of the syringe to facilitate the detaching of the syringe from the needle when it is correctly placed in the sinus. The infant is wrapped in a sheet and held on its back on the table by one nurse, while a second sits on a chair holding the head securely in the middle line and slightly flexed. The operator stands at the side of the infant, facing it, the bevel of the needle being held parallel to the skin, so that the opening in the needle will be patent with the sinus just as soon as it is entered. Before commencing, the infant is made to cry vigorously to flood the sinus with blood. The pressure in the sinus when the infant is crying is usually sufficient to throw a jet of blood right into the syringe, if the piston be held not too firmly. Indeed, in a vigorous infant, it is surprisingly difficult to force a solution into the sinus. To overcome this difficulty and to prevent much oozing from the puncture wound, it is our custom to have the feeding, due before the injection, omitted, and then feed the infant just as soon as the needle is shown to be in the sinus. If the blood does not enter the syringe under its own pressure, it is sucked into the syringe before an injection is made.

The slight disturbance and ease with which sinus work may be done, particularly in young infants, is shown in the following instances:

A sleeping infant, 6 days old, was taken from its cot, 60 c.c. of saline, necessitating two changes of syringes, were injected into the sinus and the infant put back to bed without awakening.

The same day a second infant was given 40 c.c. of saline solution, the infant rousing only when the needle was inserted, but at once going off to sleep for the remainder of the injection.

A third instance, showing a practical application of the method, was that in which blood for a Wassermann was obtained from a patient in one of our own offices, the mother holding the infant, and not realizing that blood had been withdrawn, as the operation was done quickly with the syringe obscured by gauze.

THE EFFECT OF TRANSFIXING THE SINUS BY THE NEEDLE. A REPORT OF FOUR CASES

In the early part of our sinus work it was thought that the infant might be endangered or some untoward symptom be caused by transfixing the sinus. In our experience the accident has happened four times, in none of which was there any clinical sign, such as cortical irritation, nor in the two cases which came to necropsy, was any anatomic sign, such as hemorrhage, visible. The first two cases occurred before a guard was used on the needle to prevent it entering too deeply. In both cases the operator was obtaining blood for a Wassermann reaction. In the second instance about 5 c.c. of cerebrospinal fluid was first drawn into the syringe, and then, withdrawing the needle along the line of entrance, 5 c.c. of blood were obtained, showing that the needle had transfixed the sinus.

Case 3 was in an infant dying of malnutrition, in which the family history suggested syphilis. The silk gauze slipped, allowing the needle to enter about ¾ inch; withdrawing along the line of entrance, blood was obtained, showing that the needle had transfixed the sinus. The infant died five hours afterward. The necropsy revealed no signs of hemorrhage, and showed no microscopic damage done to the sinus.

Case 4 was in an infant dying of miliary tuberculosis in which the whole venous circulation was markedly distended. The sinus was transfixed in two places. The necropsy, four hours afterward, showed the sinus and cerebral veins markedly distended, but an asbsence of clot or sign of injury. This case was thought to be very instructive, for, in view of the marked distention of the sinus, it was the type of case in which one would expect hemorrhage, if it ever occurred. Clinically and anatomically up to the present we have seen no bad results from transfixing the sinus.

In this paper we are able to report 100 cases in which the sinus has been used either for diagnosis or for therapeutic measures. Blood has been withdrawn for a Wassermann test in thirty-nine cases, ranging in age from birth to the oldest, 2 years and 5 months. In the latter the anterior fontanel was 1 inch across and ½ inch in the anteroposterior direction. It was quite firm, but fibrous, and presented no more difficulty in entering the sinus than one would expect from the nature of the tissue at that age. In these cases and the remaining cases the sinus was entered in one or more of three locations: first, the anterior angle of the fontanel; second, the posterior angle of the fontanel; third, along the line of the sagittal suture at a distance of ½ to 1½ inches behind the fontanel. We have been equally successful in the three locations, and think that, usually, the one has no advantage over the others. The three locations, however, give three chances for entering the sinus if any difficulty, as occasionally happens, is encountered.

THERAPEUTIC MEASURES

Diphtheritic Antitoxin.—This has been given in two instances, one being in a ward case, and the second, in an infant seen in consultation, 3 weeks old, with a bad diphtheritic throat of three days' duration, extremely ill, the infant having had no serum. It was thought that the only chance of saving the infant was by the intravenous injection of a large dose of antitoxin. Twenty-thousand units were accordingly given, by means of the sinus route, in a few minutes. This method of giving antitoxin intravenously should prove very valuable and save lives that otherwise might be lost.

Diarsenol.—It was with the view of giving the most intensive treatment for syphilitic infants that we first turned to the sinus in our search for a method of doing this easily and rapidly. Up to the present we have given fifty-two injections by this method. When one remembers the areas of necrosis following accidental subcutaneous injection of diarsenol or salvarsan, this fact will be apparent, that the injection is a good test of the efficiency of the sinus in therapeutic treatments. No cases have occurred in which irritation has followed the injection of the arsenical salts. In one instance adhesions or thrombosis may have occurred, although our only ground for suspecting this was the difficulty experienced in entering the sinus after six injections had been given. This was the first case in which we had given the diarsenol this way and our difficulty may have been due to lack of experience. The case went on to complete recovery, however, with repeated negative Wassermann tests. In all our other cases we have had no difficulty in giving repeated doses, so that we feel justified in concluding that thrombosis practically never occurs in the sinus as it does occasionally in the small veins following the injections.

Saline Injections.—Having in view the use of the sinus for transfusing purposes, four infants were given a saline solution, using the 20 c.c. syringe to see if any difficulty would arise while changing the syringes with the needle in the sinus. It was found by putting petrolatum on the top of the syringe, and with an assistant preventing the needle from turning by means of an artery forceps, that the syringes could be removed and replaced without any difficulty. Judging, from these saline cases, the ease and rapidity with which the solution was injected, there is no doubt that an infant could be given 100 c.c. or more of blood in a few minutes. The most interesting and practical point is that this could be carried out at the bedside and with only a few minutes' preparation. To emphasize the ease and rapidity of an injection, it may be added that four injections of diarsenol have been given by one of us in fifteen minutes, this including the preparation of each individual dose, the preparations of the infants, etc. Anyone who

has worked with the veins in infants will appreciate the importance of this statement as an evidence of the value of this procedure as a time saver.

Blood Culture. — Three cases for blood culture by the Rosenau method were carried out. There was no difficulty in getting sufficient blood, the parts being easily prepared and rendered aseptic. We have in mind the use of the sinus for giving sodium bicarbonate solution and for other therapeutic measures, but owing to being short handed and having had extra work thrown on us, we are unable to report these cases at present.

THE PRODROMAL SYMPTOMS OF INFANTILE PARALYSIS

BASED ON A STUDY OF 400 HISTORIES OF PATIENTS ADMITTED TO THE WILLARD PARKER HOSPITAL FROM JULY 1 TO SEPTEMBER 1, 1916, INCLUSIVE *

MAY G. WILSON, M.D.
NEW YORK

These histories were obtained by personal interviews with parents, corroborated, when possible, by family physicians. Every effort was made to obtain an accurate history of the onset and course of the illness prior to admission. The wide publicity given this disease resulted in the early recognition and unusually close observation of the illness and symptoms of the child.

The symptoms included in this report were those noted from the onset of illness until the appearance of paralysis. Falls, overexertion, unusual excitement, overeating, and dentition preceding the onset were given as causes. The onset as a rule, was acute, attacking an apparently healthy child unawares.

I will take up the prominent prodromal symptoms in the following order: Fever, gastro-intestinal symptoms, respiratory symptoms, nervous symptoms, urinary system, skin.

Fever.—Fever was the most constant initial symptom, being noted in 334 cases. Only 2 per cent., on careful investigation, gave no history of fever, the temperature rapidly reaching its fastigium in twenty-four to forty-eight hours. The highest temperature noted was 106 degrees, the average, 103; duration, from one to ten days, average duration, four days, falling by crisis or lysis.

The following tabulation and Chart 1 show the relation of the duration of the fever to the day of the disease.

Fever	Days	Cases
0	..	8
..	1	17
..	2	45
..	3	71
..	4	63
..	5	39
..	6	27
..	7	21
..	8	6
..	9	2
..	10	4
..	11	1

* Submitted for publication April 9, 1917.
* Read before the New York Academy of Medicine, Pediatric Section, Oct. 12, 1916.

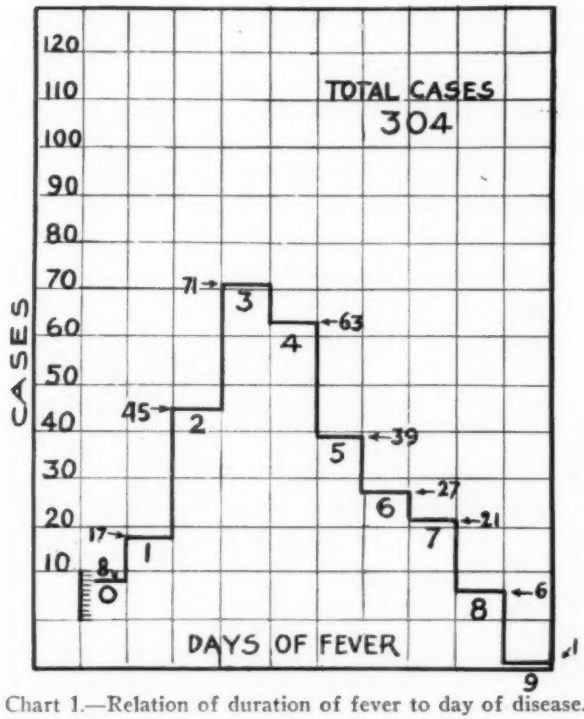

Chart 1.—Relation of duration of fever to day of disease.

In cases of remission or relapse, an initial fever of one or two days was followed by apparent health from two to six days, with a secondary fever, and ensuing paralysis.

The following tabulation and Chart 2 show the relation of the onset of paralysis to the day of the disease.

Paralysis	Days	Cases
0	..	6
..	1	10
..	2	67
..	3	120
..	4	79
..	5	27
..	6	12
..	7	10
..	8	1
..	9	1

Gastro-Intestinal Symptoms.—Vomiting: This was noted as an initial symptom in sixty-seven cases, as an early symptom in 132, sometimes occurring after the child had retired in apparently good health and slept a while; more usually, however, immediately on taking food. The vomiting was seldom repeated; in one instance it was of a projectile character.

Constipation: One hundred and fifty-six cases gave a definite history of persistent constipation for two or more days, resisting ordinary

catharsis, and only relieved by repeated enemas. Fecal scybali were often found on examination.

Diarrhea: Diarrhea was not a common symptom in this series, being present in only twenty-five cases, and being neither severe nor characteristic. Abdominal pain was noted as an initial symptom in twenty-one cases, as an early symptom in twenty-five. When present, it was usually severe, persisting several days, referred to the epigastrium or general, and in two instances simulating appendicitis.

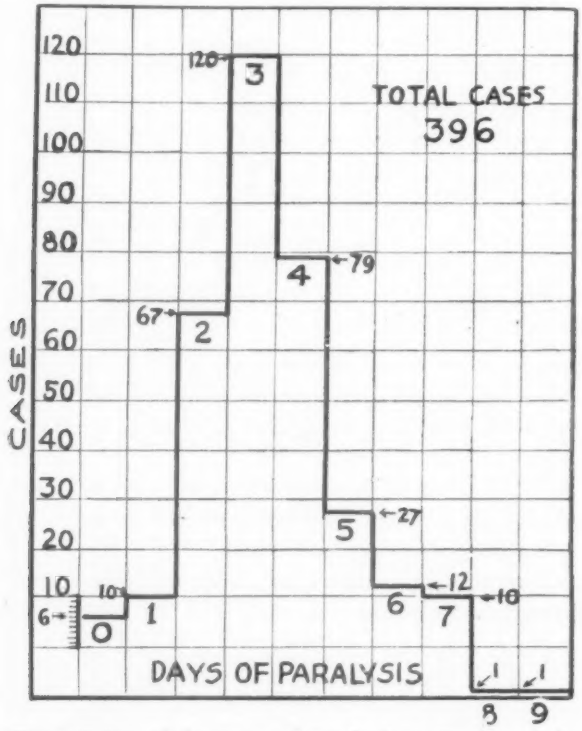

Chart 2.—The relation of the onset of paralysis to the day of the disease.

Respiratory Symptoms. — Throat: As an initial symptom, sore throat was complained of in 21 cases. A red throat was noted in 27 cases, follicular tonsillitis in 14. On examination of 100 cases on admission, injected fauces were found in 31 cases, enlarged inflamed tonsils in 11, exudate in 3, mucopurulent, frothy discharge in 7.

Epistaxis was present in two cases as an initial symptom.

Coryza was present in seventeen cases.

Conjuctivitis was present in nine cases.

Cough was present in thirty-eight cases, usually slight; two instances of severe bronchitis were noted. As a group, these symptoms were not common or characteristic.

Nervous Symptoms. — Drowsiness: Persistent drowsiness was an early and characteristic symptom, noted in 288 cases; that is, 72 per cent., varying from slight apathy to stupor in forty-seven cases. As one mother stated: "The baby just wanted to sleep all the time, and I had to keep him awake to nurse him." It was of common occurrence in the admitting room to find children sound asleep and difficult to arouse, in spite of their strange surroundings and long ride in the ambulance.

Irritability was next in frequency, noted in 153 cases. Associated with this symptom was marked hyperesthesia, noted in ninety-seven cases, the slightest touch or even approach being resented. "My baby is sore all over," said a mother.

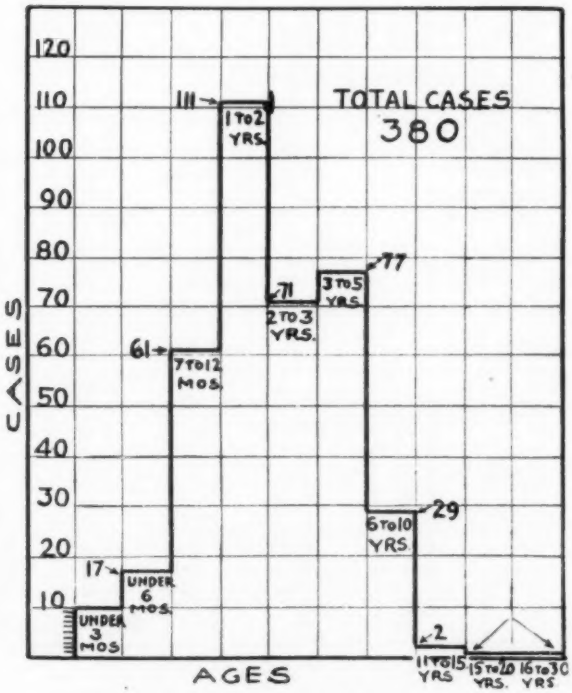

Chart 3.—Age incidence.

Tenderness and stiffness of the neck was an early and common symptom, noted in 161 cases. Retraction of the neck and flexion of the lower extremities appeared in forty cases.

Pain was present in 130 cases, usually referred to extremities, neck, back, shoulders and chest.

Tremor was noted in 113 cases, sometimes limited to a single muscle or group of muscles, usually of the extremities. Parents would remark: "Child seemed to shiver," or, "I first noticed when Mary took a piece

of bread how her hands trembled like an old man's." Tremor persisted during the febrile period, preceding the paralysis by twenty-four to forty-eight hours. Two cases showed a marked, coarse tremor, persisting for several weeks, limited to one side of the body, resembling intention tremor, absent when asleep and recurring on the slightest irritation.

Twitching was noted in sixty-four cases, sometimes choreiform. Twitching of the corner of the mouth often preceded a facial paralysis.

Headache was present in seventy-eight cases, often persistent and severe; frontal or general headache was the first symptom complained

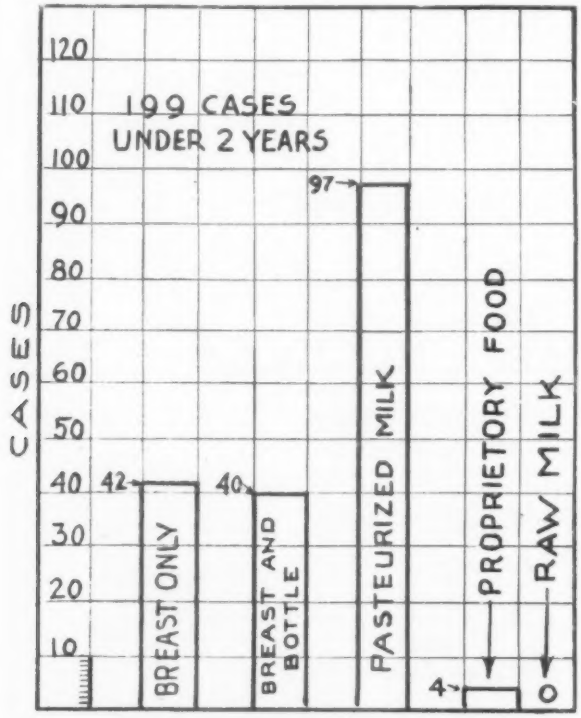

Chart 4.—Previous feeding.

of in twelve cases. Convulsions were present in six cases, as an initial symptom in three. Two cases were of children giving histories of previous convulsions.

Delirium was noted in ten cases.

Urinary System.—Twenty-one cases gave a history of some urinary disturbance, usually minor retention.

Skin.—There was noted profuse sweating in forty-five cases, and out of proportion to the fever present, usually preceding the paralysis, and as a rule, general; in a few instances, localized.

Rash: Red blotches on extremities, 2 cases; general erythema, 4 cases; macular, resembling measles, 8 cases; urticaria, 4 cases; pustular, 2 cases; herpes, 7 cases, over back and trunk, and in one case limited to arm later paralyzed.

The clinical picture of the abortive type of the disease corresponds in general with the initial stage of a typical case followed by paralysis. Mild initial symptoms may be followed by extensive paralysis, and, on the other hand, cases with severe and alarming onset have shown slight paralysis and rapid recovery.

In this series the ages were (Chart 3):

Under 3 months, 10.	3 to 5 years, 77.
7 to 12 months, 61.	6 to 10 years, 29.
1 to 2 years, 111.	11 to 15 years, 2.
2 to 3 years, 71.	16 to 30 years, 2.

Sex: 222 males and 178 females.

The race incidence was as follows:

Hebrew, 152	American, 76	Italian, 70
Irish, 50	Polish, 17	German, 6
Colored, 6	Swedish, 4	Japanese, 1

Previous contagious diseases:

Measles, 45	Scarlet fever, 6	Diphtheria, 4
Chickenpox, 4	Whooping cough, 7	

Twelve cases had had recent operations on the tonsils, 8 had hypertrophied tonsils and 87 had normal tonsils.

Of 537 exposures in the families in which the disease occurred there were 50 secondary cases.

Of the previous feeding it was ascertained as follows (Chart 4):

	Cases
Breast only	42
Breast and bottle (pasteurized milk) (Grades A and B)	40
Breast and bottle (condensed milk)	3
Bottle only (pasteurized milk) (Grades A and B)	97
Bottle only (condensed milk)	2
Bottle only (malted milk)	2
Bottle only (Nestle's food)	1

As to summer errors of diet, that is, ice cream, soda water, candy, pastries, watermelon, spoiled fruits, 163 out of a total of 292 children over 2 years old gave a history of over indulgence in this respect.

REPORT OF CASES

The following case histories illustrate the various symptoms of onset:

CASE 1.—Girl, aged 27 months; onset, Saturday, August 12, with high fever. Vomited breakfast; increasing drowsiness; bowels constipated; twitching of the corner of the mouth. Sunday, a. m., twenty-four hours after onset, facial paralysis was noted. Monday, p. m., temperature normal; child well except for facial paralysis. Discharged October 14 with slight facial paralysis.

CASE 2.—Girl, aged 7½ years; onset, Thursday, July 27, with severe frontal headache. Vomited breakfast; temperature 105. The child was extremely irritable, complained of pain in the extremities, stiffness of the neck and general twitching. Sunday, July 30, the fourth day of the disease, paralysis of legs and arms was observed. Discharged October 1, with paralysis of legs and arms.

CASE 3.—Girl, aged 2½, sister of the patient, Case 2. Gave the following history: For the previous week the child complained of general malaise, headache and abdominal pain. July 31, the child was up and about. August 1, took to bed with high fever, headache and abdominal pain. Vomited. August 3 and 4, became increasingly drowsy, going into coma. August 5. Admitted August 6, with temperature 103. August 17, normal temperature. Child fully recovered; no paralysis. Discharged October 1.

CASE 4.—Boy, aged 3, one of twins. Onset Wednesday, August 10. Wednesday, August 10, the child was playing as usual, but spent a restless night with high fever. Thursday the child was unusually sleepy and remained in bed. The bowels were constipated; complained of headache, but did not seem ill. On Saturday morning, except for the bowels not moving, the child seemed much better. Saturday, p. m., there was a marked change; twitching of the hands, a sudden difficulty in breathing and rattling in the chest. The child was admitted to the hospital at 6 p. m. and died in three hours of respiratory paralysis.

CASE 5.—Boy, one of twins, aged 20 months. (Brother died after three days' illness, July 9.) July 10, the patient had high fever, tonsillitis, constipation; child became increasingly drowsy; was irritable and did not want to be taken up. July 12, admitted to hospital; sore throat, fever, drowsy. July 14, two days later, the child was well, but very weak. July 15, third day, the child was apparently normal and was almost considered as a "no case" by one of the attending physicians who had not seen the child before.

CASE 6.—Boy, aged 20 months (brother, 3 years of age, died after an illness, July 10 to July 13, of respiratory paralysis). Onset, July 17; first symptoms vomiting, fever, increasing drowsiness, profuse sweating. July 19, mother noticed the child could not lift the upper lids of both eyes. Admitted July 20; temperature 100.5, pulse 110, respiration 30; stuporous; erythematous rash over body. The only paralysis noted was that of both upper lids. July 26, the child was standing in his crib, apparently well, but not being able to lift both eyelids.

CASE 7.—Woman, married, aged 28. Onset July 31; severe frontal headache, fever; vomited; had backache and abdominal pain and some cough. She thought she had caught cold. She took to bed August 1; sweat profusely, the symptoms of onset persisting. August 2, felt weakness in the right leg; by the next day it affected her left leg and she was unable to move it. August 3, sharp pains in both lower extremities; especially painful to the touch in the course of the nerves. On admission, August 4, there was paralysis of both lower extremities.

CASE 8.—Boy, aged 3½ years. Onset August 8. First symptom, headache; vomited. Was better the next day and was up and about. August 10, he became apathetic; had projectile vomiting all day; complained of pain in the head. Became increasingly drowsy; could not stand or sit up; was very sensitive to touch; had some stiffness of the neck and back. Admitted August 11; temperature 101; stuporous; eyes did not coordinate; muscular weakness of right and left deltoid and right and left quadriceps extensors. All reflexes absent. The child was seen January 1; no residual paralysis.

CASE 9.—Girl, colored, aged 4 years. Onset August 6, with fever, difficulty in breathing, increasing drowsiness, obstinate constipation. On the second day, twitching of extremities; cried out with pain in the head; was very sensitive to touch and had some cough. On the third day, weakness of the right and left deltoid. Admission August 9; temperature 102, pulse 140, respiration 38; throat

slightly congested; sensorium cloudy; paralysis of upper extremity and intercostals, with weakness of lower extremity; all reflexes exaggerated. The child died August 17 of respiratory paralysis.

CASE 10.—Boy, aged 7 years. Onset August 3; first symptoms, fever, vomiting, abdominal pain. Persistent headache; increasing drowsiness. August 6, patient became delirious and got out of bed. Admitted August 8. Throat congested; chest clear; abdomen tender and distended; sensorium cloudy; no definite paralysis noted; all reflexes exaggerated; spinal sign present. August 9, patient semistuporous; scaphoid abdomen; retention of urine. August 11, weakness of both deltoids; reflexes absent. August 12, general condition very much improved. August 13, paralysis of both deltoids. August 17, general condition normal; paralysis of both deltoids present.

CASE 11.—Girl, aged 7 years. Onset July 26, with fever; vomited several times; was extremely irritable and did not wish to be handled; complained of headache and pain in the throat. July 27, sweat profusely; was drowsy and voice became hoarse; had difficulty in swallowing. Admitted to the hospital, July 28; temperature 101; paralysis of the muscles of deglutition and laryngeal involvement. Died July 30.

CONCLUSION

The prodromal period has been found to be the most important stage in the course of the disease, both as to early quarantine and treatment. A careful history, while not diagnostic, is very suggestive, particularly in an epidemic.

I wish to express my thanks to the director, visiting and resident staff of the Willard Parker Hospital for the opportunity and cooperation afforded me in this study.

601 West 177th St.

POSTERIOR BASIC (WALLED-OFF) MENINGITIS. REPORT OF A CASE WITH RECOVERY*

ABRAHAM SOPHIAN, M.D.
KANSAS CITY, MO.

Recovery after the development of "walled-off" meningitis is rare. Fisher[1] reported the first case recovery in a child. Verbizer and Chauvel[2] report recovery in an adult.

REPORT OF CASE

History.—A child, 11 months old, a patient of Dr. Saulberry, developed meningococcic meningitis. Serum treatment was begun late, two weeks after the onset, and was discontinued in the presence of a severe, persistent hydrocephalus, owing to objection by the parents. Four weeks after the onset the child presented the classical picture of "basic" meningitis; opisthotonos was marked, the eyes were wide open, staring, with lids retracted, the face expressionless. Hydrocephalus was extreme; head large, fontanel bulging, sutures widely separated. The heart's action was very irregular; respirations were irregular, of the Biot type. There was persistent tonic spasm of the extremities, with occasional clonic convulsions. Vomiting was repeated and projectile. The temperature reached 102 F., and was of irregular type.

Diagnosis.—Lumbar puncture at two levels yielded a few drops of clear, sterile cerebrospinal fluid. The clinical picture of hydrocephalus, meningeal irritation and absence of fluid in the subarachnoid space pointed to an encapsulated (walled-off) hydrocephalus, due to a closure of the foramina of Key, Retzius and Majendie.

Management.—Ventricular puncture was indicated. An ordinary lumbar puncture needle, with stylet attached, was introduced into the right ventricle through the extreme lateral border of the anterior fontanel. The needle was directed downward, backward and inward, and the ventricle was reached at a depth of about 2 inches. Fifty cubic centimeters of slightly turbid fluid under very high pressure were removed and 20 c.c. of serum injected. There was a fall of 10 mm. Hg in blood pressure on the removal of the fluid, but no change on injecting the serum. The left ventricle was tapped the next day, 80 c.c. of turbid fluid removed and 20 c.c. of serum injected, with blood pressure change as on the previous day. The first fluid showed many pus cells and a few meningococci, extracellular and intracellular; the second specimen had only a few pus cells and intracellular organisms.

The right ventricle was tapped forty-eight hours later and 60 c.c. of clear fluid were removed. No meningococci were found. There was marked clinical improvement. The parents at this stage decided against any further intracerebral treatment. Improvement continued, however, and two weeks later the child had apparently recovered completely, with no obvious sequelae.

Subsequent Course.—Two months later the child presented a most unusual picture. He was bright, and had grown tremendously, having put on in two months the equivalent growth of a year and a half. He was active, playful,

* Submitted for publication April 14, 1917.

1. Fisher: New York Med. Jour., March 26, 1910.
2. Verbizer and Chauvel: Bull. et mém. Soc. méd. d. hôp. de Paris, 1916, **32.**

could see well, and had developed mentally almost in proportion to his skeletal growth. There was a large thymus. The head was large. During the previous month, however, the child had developed alarming spells of unconsciousness, marked cyanosis and dyspnea, lasting a few minutes; at first occurring every few days, then several times daily. I interpreted the condition as being due to a persistent hydrocephalus, and attributed the excessive skeletal development to pituitary involvement.

Lumbar puncture yielded 60 c.c. of clear sterile fluid under high pressure. The puncture proved that the communication between the ventricles and subarachnoid space had been reestablished. There were no convulsions for three days, and during the following two weeks convulsions occurred at infrequent intervals. Thyroid extract was administered.

A year later I learned that the child had grown very stout and large for his age, but was somewhat subnormal mentally. The attacks of cyanosis ceased a few months after the last puncture.

This case proved not only that recovery is possible, but that the communication between ventricles and subarachnoid space can be reestablished.

Various methods may be used to drain the ventricles. The simplest is puncture through the anterior fontanel, with or without drainage by tube or silk passing from without into the ventricles.[3] When the fontanel is closed, Kocher's point for trephining over the anterior fontanel, or Keene's point for the posterior horn of the ventricle, may be selected. Haynes'[4] cisternasinus drainage is intended to direct the cerebrospinal fluid into one of the brain sinuses. Sharp[5] describes the treatment of internal hydrocephalus by introducing linen strands into the ventricle and bringing the ends out through the temporal muscle and fascia, obtaining drainage in this way. Haynes' and Sharpe's methods are only applicable in that type of basic meningitis with sterile ventricular cerebrospinal fluid.

The most important requisites for successful results are early diagnosis and immediate intraventricular treatment. More important, however, is the prevention of this complication of simple epidemic meningitis. The secret is early diagnosis, early and persistent intraspinal serum treatment, and control of hydrocephalus.

Holmes and Twenty-Third Street.

3. Sophian, A.: Epidemic Meningitis, St. Louis, 1913.
4. Haynes: New York State Jour. Med., 1916, **16,** 174.
5. Sharp: Am. Jour. Med. Sc., April, 1917.

PROGRESS IN PEDIATRICS

REVIEW OF OPHTHALMOLOGY*

HOWARD F. HANSELL, M.D.
PHILADELPHIA

Amaurotic Family Idiocy.—Amaurotic family idiocy is a disease of the central (cerebral) nervous system in which the principal symptoms consist in gradually increasing physical weakness, arrested mental development and gradual loss of sight. It is found almost exclusively in Jewish children 2 or 3 years of age, although a few cases have been reported in children of other nationalities.

Ophthalmoscopic examination shows a whitening optic nerve and a deep red round spot in the fovea, surrounded by a whitish patch of retinal degeneration. The disease is fatal in the course of a few months.

The clinical similarity of amaurotic family idiocy to "cerebral family degeneration" has led to the belief that they are identical affections, and that the latter title is given to the affection in its later stages. Batten and Mayou[1] say:

> There is a close relationship between these cases (family cerebral degeneration) and the cases of amaurotic idiocy. The only difference in the two diseases appears to be that the macular region in amaurotic family idiocy shows a much more marked edema of the internuclear layers, and therefore, a white area around the macula which is associated with a hole or thinning of the retina at the fovea, due to edema.

The late George Coats[2] dissents from this description of the histology of the retina in amaurotic family idiocy. He says that the changes are absolutely confined to the ganglion cell and nerve fiber layers; that there is no edema, folding, thinning, or formation of holes at the fovea. The pathology of family cerebral degeneration is: (1) Certain cytologic degenerations of the ganglion cells; (2) a diminution amounting in places to a complete disappearance of those elements; (3) edema of the internuclear layer; (4) loss of rods and cones and of the outer nuclear layers; (5) thickening of the supporting elements; (6) immigration of pigment into the outer layers of the retina.

Another point brought out by Mr. Coats, observed and commented on by other observers, is that sudden local obstruction of the circula-

* Submitted for publication Feb. 25, 1917.

1. Batten and Mayou: Proc. Roy. Soc. Med., 1915, **8,** Ophth. Sect.
2. Coats: Ophthalmoscope, April, 1916.

tion, as in thrombus of the central artery of the retina, does not produce edema of the neighboring tissues, or the tissues previously supplied by the artery, as has been until recently held, but an acute coagulation necrosis. In support of this view, he says:

> 1. Nowhere in the body does obstruction of an artery cause edema. The changes are a white infarct of the inner layers, which receive their supply from the central artery. 2. The ophthalmoscopic appearances produced by macular edema are well known and may be studied in cases of nephritis and papilledema. They differ in that the opacity is never nearly so dense and there is no cherry spot. 3. The distribution of the opacity is at variance with the hypothesis that it results from edema. If due to that cause the opacity should be present in the periphery as well as in the macula. 4. Edema offers no explanation of the fact that the opacity always persists for about the same time—the period required for the breaking down and absorption of the necrotic layers, not for the absorption of the edema, for it is certain that the rapidity and completeness of the restoration of the circulation is subject to very large variations.

Congenital Glaucoma and Some Allied Conditions.—This disease is better known as buphthalmos or hydrophthalmus.[3] It is, although not always recognized in infancy, congenital in origin, and dependent on defective development of the parts concerned in excretion; bilateral in 63 per cent. of the cases, and twice as common among boys as among girls. The symptoms are slow but steady enlargement of the eye; hemispherical or globular cornea; greatly deepened anterior chamber; uniformly distended sclera; tremulous iris; increase of intra-ocular pressure; cupping of the optic disk; marked impairment of vision. The affection is an arrest of development and is due to obstruction of excretion (rather than the result of inflammation) through closure or absence of the filtration angle in the anterior chamber. He advises trephining as the most satisfactory surgical cure, and adds that we must never forget the possibility that congenital syphilis may be playing a part in any case of buphthalmos, and that we must consequently order our methods of diagnosis and our therapy in the light of this knowledge.

Interstitial Keratitis.—Dr. George F. Suker,[4] in a discussion of the use of salvarsan and neosalvarsan in the treatment of interstitial keratitis, says that it makes no difference whether antispecific treatment is employed or not, as the patient will improve under general treatment. He modifies this statement, however, by admitting that salvarsan is of value when the spirochetes are present in the corneal tissue proper. When a local reaction, as shown by circumcorneal injection, follows the initial dose of salvarsan the spirochetes are frequently found in the corneal stroma. Therefore, salvarsan acts somewhat after the manner of tuberculin, and in such cases a marked improvement follows its use.

3. Elliot, R. H.: Ophth. Rec., November, 1916.
4. Suker, G. F.: Ophth. Rec., November, 1916.

Myopia.—Increase in length of the anteroposterior axis of the eyeball is rarely congenital and seldom found in children under 10 years of age. But that myopia does exist at an early age is shown by reports by Harman,[5] Snell[6] and Walker.[7] Tenner[8] notes that myopia, whether present or not at the beginning of school life, may develop and gradually increase with age so that at 16 years refraction of the ball has increased in nearly one-half of the children who have inherited a tendency to myopia.

The correction of hyperopia and astigmatism would prevent the stretching of the sclera, and hence myopia, in many instances. In addition to the careful correction of refraction under paralysis of accommodation, Crampton[9] says the attention of the guardian or teacher should be directed to other aids, such as the correction of faulty habits, including excessive use of the eyes for near work; tendency to stooping, and the proper adaptation of desks to the size of the student and the source of illumination.

Trachoma.—The surgical treatment of trachoma, the scourge of every country and every people, has been the subject of many recent papers, and each writer seems to believe that the method employed by him is the only one and that will effect a cure in most cases. Despite the laboratory investigations, no progress has been made in the discovery of the cause, and until the etiology is better understood the value of any treatment must remain problematic.

The primary objects of treatment are, according to Wright,[10] to minimize conjunctival hypertrophy; to produce prompt absorption or removal of existing granulations; to prevent extensive destruction of the conjunctival mucosa; to limit secretion and guard against complications which seriously endanger corneal integrity; to restore the damaged structures, and to prevent deformity and impairment of vision.

The surgery embraces expression by manual manipulation or by forceps; vigorous rubbing of the conjunctiva with 1 to 5,000 mercuric chlorid solution; the subcutaneous injection of the contents of the trachoma follicles triturated in a saline solution; grattage; rolling and rubbing the bleeding surface with gauze saturated with a solution of mercuric cyanid; expression, followed by resection of the tarsal conjunctiva; electrolysis; removal of the tarsal cartilage and palpebral conjunctiva; radium; the Finsen ray.

5. Harman: Proc. Roy. Soc. Med., Nov. 4, 1914, Ophth. Sect.
6. Snell: Ophth. Rev., 1915, **34,** 95.
7. Walker: Ophth. Rev., 1915, **34,** 191.
8. Tenner: New York Med. Jour., 1915, **102,** 611.
9. Crampton: Am. Jour. Ophth., 1915, **32,** 86.
10. Wright: Internat. Clin., Series 25, 1915, **3,** 62.

The latest medicinal or bacteriologic remedy consists in the use of tablets containing the Bulgarian bacillus. C. R. Hudgel[11] reports the following case:

> A patient with trachoma in the second stage came under my care. I reduced the tablets to a fine powder, everted and scarified the lids and dusted the powder on the lids night and morning. The pannus disappeared and in two weeks the patient was well.

Daggett (*Amer. Jour. Clin. Med.*, July, 1913) also has had success with the Bulgarian bacillus.

Crawford of Nashville used this remedy in fourteen cases and claims surprisingly good results.

Eye Signs of Poliomyelitis.—During the epidemic of poliomyelitis of 1916, no writer has, as far as it is possible to learn, laid much stress on eye signs or symptoms, or pointed out that the involvement of the eyes might indicate the presence of this affection rather than other forms of disease of the cerebrospinal system. The value of eye signs in determining the diagnosis may accordingly be regarded as negative.

An isolated extra-ocular paralysis or even a monocular or binocular optic neuritis are not significant of any definite lesion, and are found in many diseases of the cerebrospinal system. As C. H. Fife[12] says: "An infectious disease that may involve practically any portion of the cerebrospinal axis necessarily presents a varied and kaleidoscopic symptomatology." In the lengthy and able discussion following the reading of his paper the only mention of ocular involvement was made by T. H. Weisenburg, who had studied about six hundred cases of poliomyelitis admitted to the Municipal Hospital of Philadelphia. He said:

> There have been a large number of bulbar cases; in these the seventh nerve is generally implicated, although in my own examinations I found, as a rule, an associated paralysis of the sixth and fifth nerves. Paralysis of the ocular group is very uncommon. Blindness I have seen only once.

Posey[13] reports one case of primary double optic nerve atrophy, one of paralysis of the sixth and seventh nerves, one of double ptosis (he expressed himself as uncertain as to whether the ptosis had not antedated the attack of poliomyelitis), and one of complete external ophthalmoplegia. As to the origin of the ocular paralysis, Weisenburg says:

> I am in doubt as to the meningeal origin of the optic nerve atrophy or muscular palsies in infantile paralysis. I have failed to detect what I would consider either an anterior or posterior root lesion, or a cranial nerve lesion due to meningitis, for while the pathology of the disease is undoubtedly a meningomyelitis, yet the meningeal lesions are not sufficient to cause pathologic manifestations for any length of time.

---

11. Hudgel: Northwest Med., June, 1914.
12. Fife: Pennsylvania Med. Jour., November, 1916.
13. Posey: Ophth. Rec., December, 1916.

Ethylhydrocuprein.—Among the newer remedies which has proved its worth in a large number of clinical trials is ethylhydrocuprein, a derivative of quinin. Its name is misleading, for unless one knows its family connections one is apt to believe it to be a form of copper. This error is not infrequent. It is also known as "optochin." Its special prerogative is inflammation due to the presence of the pneumococcus, although it has been recommended when the affection is due to the staphylococcus, the streptococcus, the gonococcus, and to mixed infections. One of the earliest papers to appear was that by Puscairu.[14] In her experience in the treatment of eight patients of gonorrheal ophthalmia "It was found that with this treatment absolutely no irritation or intoxicative symptoms were manifested, and all the cases were clinically cured long before the gonococci disappeared from the smears, so that only a bacteriologic examination could give positive information." Wyler[15] reported on two cases which are of sufficient interest to warrant more than mention: 1. An infant, 7 days old, three days after delivery developed ophthalmia. Treatment: frequent flushing with permanganate 1 to 10,000, later chlorin water, 1 dram to 6 ounces, and instillation of ethylhydrocuprein (optochin) hourly. In ten days the baby opened its eyes, the corneas were clear and "everything as it should be." On the twelfth day the corneas became cloudy and ulcerated and, in spite of every effort, perforated. 2. In a severe type of gonorrheal ophthalmia in a boy, 19 months old, limited to one eye. The cornea became opaque, but did not perforate. He reports two cases of mild vernal catarrh, the symptoms of which were greatly ameliorated by instillations of ethylhydrocuprein. This is a valuable observation, since all other remedies have proved practically inefficacious, except for the transient relief from cocain and epinephrin. H. Davier[16] says that ethylhydrocuprein has a direct chemotherapeutic action on the pneumococcus which is due to a bacteriotropism. He reported eight cases of corneal ulceration, seven of which were promptly cured. Wiener[17] healed a hypopion ulcer in the course of two days by applying the remedy for half a minute every hour for six hours on the first day and for twelve hours on the second day.

In pneumococcic ulcers of the cornea ethylhydrocuprein is said to be a specific. Ramsay[18] treated with success thirty-four patients with a 1 per cent. or 2 per cent. aqueous solution applied as follows: After the floor of the ulcer had been cleansed with a quinin or chinosol lotion

14. Puscariu: Klin. Monatsbl. f. Augenh., 1914, **53,** 342; Ibid., Clin. opht., 1914, **20,** 357.
15. Wyler: Ann. Ophth., January, 1916.
16. Davier: Clin. opht., January, 1916.
17. Wiener: Med. Rec., New York, Jan. 17, 1914.
18. Ramsay: Glasgow Med. Jour., May, 1915.

the boundaries of the ulcer are defined by fluorescein. A dossel of wool saturated with a 1 per cent. solution of ethylhydrocuprein is kept in firm contact with the ulcer for about two minutes, and a 1 per cent. solution instilled frequently. Kümmel[19] reports seventeen cases of ulcus serpens thus treated, which showed clearly the good effects of the remedy, only one case proving obstinate. Goldschmidt,[20] Stengele[21] and Holth[22] report excellent results in pneumococcic affections. Hansell[23] had phenomenal success in a case of pure pneumococcic ulceration in a patient 3 years of age. The remedy has been employed in suppurative dacryocystitis with good results by Stengele[24] and Posey.[25] Weeks, in a paper read before the American Ophthalmological Society, May, 1916, warns against the indiscriminate use of ethylhydrocuprein internally. Since it is strongly related to quinin its use may cause sudden blindness; he reports such a case.

Vernal conjunctivitis, also designated by various other names, such as hyperplastic conjunctivitis; fibroma of the limbus; phlyctenula pallida, and vernal catarrh, is not uncommon in the warm months. It attacks principally young persons from 10 to 20 years of age, preferably males, recurs for several years, with entire relief in the cold months, and is attended with intolerable itching. Many remedies have been tried and unsuccessfully; to quote de Schweinitz,[26] "weak astringents and antiseptic lotions; the application of boroglycerid to the everted lid; the systematic use of a preparation of suprarenal extract or of adrenalin chlorid 1 to 10,000; sulphate of quinin; watery solution of ichthyol (1 to 2 per cent.); salicylic ointment, 1 per cent.; massage with yellow oxid ointment; electrolysis; a collyrium of antipyrin (10 per cent.); instillations of a 2 per cent. protargol; Roentgen rays; radium, and the internal administration of arsenic."

It may be inferred that no remedy has been satisfactory. If the etiology were known the benefit of treatment would be more assured. The writer tried ethylhydrocuprein in the case of a young woman who suffered almost intolerably each summer for several years from itching of the lids. She used a 2 per cent. solution, afterward weakened to 1 per cent., three or four times a day, and with this remedy reported that the itching was well controlled.

Wyler[15] reports two cases: Dr. Z., used a 1 per cent. instillation every two hours. "At the end of a week the patient reported absolute comfort

19. Kümmel: München. med. Wchnschr., 1914, **61,** 1326.
20. Goldschmidt: München. med. Wchnschr., 1914, **61,** 1505.
21. Stengele: Klin. Monatsbl. f. Augenh., 1915, **54,** 446.
22. Holth: Wchnschr. f. Therap. u. Hyg. d. Auges, 1914, **17,** 295.
23. Hansell, H. F.: Ophth. Rec., March, 1916.
24. Stengele: Ann. d'oculist, August, 1914.
25. Posey, W. C.: Ophth. Rec., June, 1916.
26. De Schweinitz: Diseases of the Eye, Ed. 8, 1916.

from the itching, and he has continued to use this preparation when necessary through the entire summer with perfect relief." The second patient received the same treatment with the same result.

Vernal catarrh may be confused with hay-fever, rose cold and similar acute nasal affections, but has many points of difference, such as duration, gradual onset and decline, and the flat epithelial outgrowths from the conjunctiva that have not been described as having been associated with the nasal affection. The remedy is harmless, and easily applied.

1528 Walnut Street.

CURRENT PEDIATRIC LITERATURE

ANATOMY AND PHYSIOLOGY

Thymus, Functions of.—Y. Tongu.
Mitt. a. d. med. Fakultät. d. k. Univ. Tokyo, 1917, **16,** 539.

Urine, Nitrogen Partion of, in Normal Children.—M. R. Johnston and B. S. Veeder.
Am. Jour. Dis. Child., 1917, **13,** 404.

BACTERIOLOGY AND PATHOLOGY

Carcinomatous Ovarian Teratoma with Premature Puberty and Precocious Somatic Development.—R. H. Harris.
Surg., Gynec. and Obst., 1917, **24,** 604.

Glandular Enlargements, Cervical, in Children.—I. W. Kahn.
Med. Rec., New York, 1917, **91,** 672.

Jaundice in Childhood, Acute Catarrhal.—J. A. Rawlings.
Southwestern Med., 1917, **1,** 36.

Microbic Flora in Parturient Vagina, Mouth and Rectum of New-Born.—A. Hymanson and J. J. Hertz.
Am. Jour. Obst. and Dis. Women and Child., 1917, **75,** 662.

METABOLISM AND NUTRITION

Acidosis, Blood Urea and Plasma Chlorids in Uranium Nephritis, Study of, and of Protective Action of Sodium Bicarbonate.—K. Goto.
Jour. Exper. Med., 1917, **25,** 693.

Blood, Nonprotein Nitrogen of, in Atrophic Infants.—L. D. Minsk and L. W. Sauer.
Am. Jour. Dis. Child., 1917, **13,** 397.

Feeding, Artificial, of Normal Infants.—C. V. Rice.
Oklahoma State Med. Assn. Jour., 1917, **10,** 146.

Feeding, Infant.—M. B. Springer.
Oklahoma State Med. Assn. Jour., 1917, **10,** 143.

Milk, Bacillus Coli in; Preliminary Report.—F. A. Baldwin.
Am. Jour. Pub. Health, 1917, **7,** 391.

Milk, Cow's, Gestation versus State of Lactation as Factors Causing Abnormal Composition of.—L. S. Palmer and C. H. Eckles.
Am. Jour. Dis. Child., 1917, **13,** 413.

Milk Curdling Properties of Pancreatic Juice.—H. G. Chapman and A. H. Moseley.
Med. Jour. of Australia, 1917, **1,** 223.

Milk, Skim, as Food.—J. P. Street.
Modern Hosp., 1917, **8,** 253.

Mineral Substances in Connection with Nutritional Disturbances in Infants.—C. Friderichsen.
Ugesk. f. Laeger, 1917, **79,** 446.

Pellagra in Children.—W. A. Murphy.
Arch. Pediat., 1917, **34,** 254.

Scurvy at Petrograd; 500 Cases.—A. A. Nechaeff.
Russkiy Vrach, 1916, **15,** 1216.

Scurvy, Monosymptomatic Form of, in Infant.—S. de Stefano.
Pediatria, Naples, 1917, **25,** 227.

DISEASES OF THE NEW-BORN

New-Born, Resuscitation of.—R. W. Faulk.
Southern Med. Jour., 1917, **10,** 337.

ACUTE INFECTIOUS DISEASES

Cerebrospinal Fever, Recent Results in Serum Treatment of.—A. G. Robb.
Brit. Med. Jour., 1917, **1,** 478.

Diphtheria, Isolated Nasal.—J. D. Rolleston.
Brit. Jour. Child. Dis., 1917, **14,** 21.

Diphtheritic Paralysis, Vaccine Therapy of.—S. Maggiore.
Pediatria, Naples, 1917, **25,** 193.

Infantile Paralysis Commission, Report of for 1916.—F. W. Peabody.
Boston Med. and Surg. Jour., 1917, **176,** 636.

Infections in Childhood.—W. M. Sanger.
Oklahoma State Med. Assn. Jour., 1917, **10,** 139.

Measles an Alimentary Arsenical Poisoning.—S. V. de Castro.
Semana méd., 1917, **24,** 49.

Measles from Standpoint of Prevention.—J. G. Wilson.
Arch. Pediat., 1917, **34,** 241.

Measles, Ocular Complications of.—C. A. Bahn.
New Orleans Med. and Surg. Jour., 1917, **69,** 745.

Measles, Symptoms of.—S. F. Braud.
New Orleans Med. and Surg. Jour., 1917, **69,** 739.

Meningitis, Acute Cerebrospinal.—E. Belliboni.
Gazz. d. osp., 1917, **38,** 211.

Meningitis, Cerebrospinal.—W. E. C. Dickson.
Brit. Med. Jour., 1917, **1,** 454.

Meningococcus Infection, Extrameningeal Manifestations of.—A. Ferrannini.
Riforma med., 1917, **33,** 295.

Paralysis, Infantile.—W. A. Evans.
Southern Med. Jour., 1917, **10,** 311.

Paralysis, Infantile.—M. Frank.
New Jersey Soc. Med. Jour., 1917, **14,** 147.

Paralysis, Infantile, Analysis of Preparalytic Symptoms of.—J. A. Colliver.
Arch. Pediat., 1917, **34,** 263.

Paralysis, Infantile, Recent Epidemic of.—H. Emerson.
Bull. Johns Hopkins Hosp., 1917, **28,** 131.

Pneumonia, Optochin Treatment of.—F. Vermehren.
Hospitalstidende, March 7, 1917, p. 221.

Poliomyelitis, Acute Anterior.—G. Purdy.
Kentucky Med. Jour., 1917, **15,** 158.

Poliomyelitis, Acute Anterior.—W. W. Durham.
Kentucky Med. Jour., 1917, **15,** 157.

Poliomyelitis, Acute, Medical Aspect of.—F. D. Francis.
Illinois Med. Jour., 1917, **31,** 301.

Poliomyelitis, Acute, Anatomic Study of Fifteen Cases of.—W. B. Blanton.
Jour. Med. Research, 1917, **36,** 1.

Poliomyelitis, After-Treatment of.—A. C. Geyser.
New York Med. Jour., 1917, **105,** 692.

Poliomyelitis, Cause of.—H. Greeley.
Boston Med. and Surg. Jour., 1917, **176,** 540.

Poliomyelitis, Cultivation and Immunologic Reactions of Globoid Bodies in.—H. L. Amoss.
Jour. Exper. Med., 1917, **25,** 545.

Poliomyelitis, Neutralization of Virus of, by Nasal Washings.—H. L. Amoss and E. Taylor.
Jour. Exper. Med., 1917, **25,** 507.

Poliomyelitis of Monkeys, Bacteriologic Observations in Experimental.—E. C. Rosenow and E. B. Towne.
Jour. Med. Research, 1917, **36,** 175.

Poliomyelitis, Passage of Neutralizing Substances from Blood into Cerebrospinal Fluid in.—S. Flexner and H. L. Amoss.
Jour. Exper. Med., 1917, **25,** 499.

Poliomyelitis, Pathologic Effects of Streptococci from Cases of, and Other Sources.
Jour. Exper. Med., 1917, **25,** 557.

Poliomyelitis, Relation of Meninges and Choroid Plexus to.—S. Flexner and H. L. Amoss.
Jour. Exper. Med., 1917, **25,** 525.

Poliomyelitis, Serum Treatment of Twenty-Six Cases of Epidemic.—H. L. Amoss and A. M. Chesney.
Jour. Exper. Med., 1917, **25,** 581.

Poliomyelitis, Treatment of Acute Stage of, from Neuro-Orthopedic Standpoint.—I. Strauss and P. W. Nathan.
Med. Rec., New York, 1917, **91,** 575.

Poliomyelitis Virus, Survival of, for Six Years in Glycerol.—S. Flexner and H. L. Amoss.
Jour. Exper. Med., 1917, **25,** 539.

Rabies, Paralytic Accidents After Treatment for.—C. R. Mejio.
Semana méd., 1917, **24,** 10.

Rabies, Preventive Treatment of.—F. Murillo.
Siglo méd., 1917, **64,** 166.

Scarlet Fever an Alimentary Iodin Poisoning.—S. V. de Castro.
Semana méd., 1917, **24,** 49.

Scarlet Fever, Osteomyelitis of Sinuses of Nose Complicating.—H. L. Lynah.
Laryngoscope, 1917, **27,** 176.

TUBERCULOSIS AND SYPHILIS

Adenitis, Tubercular Cervical, in Children.—W. E. Ladd.
Surg., Gynec. and Obst., 1917, **24,** 467.

Syphilis, a Family with Inherited; Five of Eight Children Require Appendicectomy.
Bull. de l'Acad. de méd., Paris, 1917, **77,** 387.

Syphilis, Molar Tooth Signs of Inherited.—R. Sabouraud.
Presse méd., 1917, **25,** 169.

Tuberculous Disease of Children.—F. C. Moor.
Southern Med. Jour., 1917, **10,** 300.

Tuberculosis of the Hip, in Children.—E. L. Hendricks.
Illinois Med. Jour., 1917, **31,** 238.

Tuberculosis, Pulmonary and Tracheobronchial, in Childhood, D'Espine's Sign a Valuable Aid in Diagnosis of.—C. J. Bloom.
New Orleans Med. and Surg. Jour., 1917, **59,** 748.

Wassermann Test, Method for Obtaining Blood from Infants and Young Children for.—A. L. Kastner.
Wisconsin Med. Jour., 1917, **15,** 430.

GASTRO-INTESTINAL SYSTEM

Anus, Imperforate.—B. P. Thom.
Med. Rec., New York, 1917, **91,** 678.

Cyclic Vomiting, with Acetonemia (Acidosis).—Nondiabetic Acetonuria and Diaceturia.—F. P. Weber.
Brit. Jour. Child. Dis., 1917, **14,** 33.

Duodenal Ulcers in Infancy.—A. Brown.
Canadian Med. Assn. Jour., 1917, **7,** 320.

Oxyuris Vermicularis in Denmark.—S. Müller.
Hospitalstidende, 1917, **60,** 297.

Peritonitis, Pneumococcic, in Infancy and Childhood.—I. A. Abt.
New York Med. Jour., 1917, **105,** 769.

Pylorus, Stenosis of, Diagnosis and Treatment of Congenital.—C. G. Grulee and Dean Lewis.
Arch. d. mal du cœur, Paris, 1917, **10,** 97.

Tongue as the Mirror of the Stomach, Especially with Achylia and Pernicious Anemia.—A. Faber.
Ugesk. f. Laeger, 1917, **79,** 409.

RESPIRATORY SYSTEM

Emphysema, Subcutaneous, in a Child—Foreign Body in Bronchi; Report of Case.—H. C. Whelchel.
Georgia Med. Assn. Jour., 1917, **6,** 249.

Foreign Bodies, Diagnosis and Removal of, from Trachea, Bronchi and Esophagus.—C. L. Pennington.
Georgia Med. Assn. Jour., 1917, **6,** 246.

BLOOD AND CIRCULATORY SYSTEM

Cardiac Diseases in Children, and Cardiac Clinic.—J. S. Ferguson.
Arch. Pediat., 1917, **34,** 269.

Endocarditis, Bacteriologic Studies in Subacute Streptococcus.—R. A. Kinsella.
Arch. Int. Med., 1917, **19,** 367.

Endocarditis, Congenital, Case of.—H. E. Tuley and J. W. Moore.
Am. Jour. Dis. Child., 1917, **13,** 426.

Endocarditis in Children, Etiology and Diagnosis of.—G. H. Withers.
Illinois Med. Jour., 1917, **31,** 263.

Heart Disease, Unusual Malformations of, in Case of Congenital.—J. I. Robertson.
Jour. Path. and Bact., 1916, **21,** 78.

Heart Failure, a Case of During Operation for Removal of Tonsils and Adenoids; Heart Massage Through Abdominal Incision; Recovery.—W. M. Mollinson.
Brit. Jour. Dis. Child., 1917, **14,** 42.

Heart Murmurs, Character of, with Congenital Cyanosis.—L. Bard.
Arch. d. mal du cœur, Paris, 1917, **10,** 97.

Hemangioma Plus Lymphangioma; Two Cases.—J. Bolognesi.
Lyon chir., 1917, **13,** 957.

Leukemia, Case of, in a Boy, with Some Observations on Benzol.—F. S. Winslow and W. D. Edwards.
New York State Jour. Med., 1917, **17,** 116.

Leukemia, Infective Theory of.—G. Ward.
Brit. Jour. Dis. Child., 1917, **14,** 10.

NERVOUS SYSTEM

Amaurotic Family Idiocy, Nine Cases of.—J. H. McHenry.
Arch. Pediat., 1917, **34,** 161.

Amaurotic Family Idiocy, Two Cases of (Tay's-Sachs' Disease).—E. F. Leonard.
Illinois Med. Jour., 1917, **31,** 327.

Amyotonia Congenita: Survey of Known Cases; Report of Three Cases.—H. K. Faber.
Am. Jour. Dis. Child., 1917, **13,** 305.

Cephalocele, Anterior; Three Cases.—M. H. Vegas and J. M. Jorge.
Rev. Soc. méd. arg., 1917, **26,** 5.

Cerebrospinal Fluid, Possible Functions of.—W. D. Halliburton.
Brain, London, 1916, **39,** 224.

Chorea, Organic Nature of Sydenham's.
Rev. Soc. méd. arg., 1917, **26,** 35.

Children Who Need Special Care, How We May Discover.—R. M. Yerkes.
Mental Hygiene, 1917, **1,** 252.

Chorea, Sydenham's, with Special Reference to Treatment.—C. F. Daniel.
Ohio State Med. Jour., 1917, **13,** 243.

Dementia Paralytica, Four Cases of Juvenile.—G. C. Bolton.
Nederl. Tijdschr. v. Geneesk., 1917, **1,** 378.

Diplegia, Cerebral, with Abnormal Flexibility (Atony or Hypotonia) of Ankle Joints.—F. P. Weber.
Brit. Jour. Dis. Child., 1917, **14,** 31.

Diplegics, Congenital Cerebrocerebellar; Their Training Treatment.—L. P. Clark.
Med. Rec., New York, 1917, **91,** 751.

Epilepsy, Clinical Studies in.—L. P. Clark.
Psychiatric Bull., 1917, **2,** New Series, p. 21.

Epilepsy, Some Unusual Features of Jacksonian.—S. Leopold.
Jour. Nerv. and Ment. Dis., 1917, **45,** 220.

Epilepsy, Case of.—F. M. Stites.
Kentucky Med. Jour., 1917, **15,** 155.

Feebleminded, Frequency and Significance of Dysfunction of Internal Secretory System in.—C. P. McCord and H. A. Haynes.
New York Med. Jour., 1917, **105,** 583.

Feeblemindedness as Seen in Court.—V. V. Anderson.
Mental Hygiene, 1917, **1,** 260.

Infundibular Tumor in a Child Causing Diabetes Insipidus with Tolerance of Alcohol.—L. Newmark.
Arch. Int. Med., 1917, **19,** 550.

Mental Tests and Test Series, Some Criteria for Evaluation of.—F. Mateer.
Mental Hygiene, 1917, **1,** 241.

Myoclonus; Report of Case of Unverricht's Myoclonus Epilepticus.—B. B. Rowley.
Wisconsin Med. Jour., 1917 **15,** 401.

Paralysis, Obstetric, Review of Its Mechanism and Outlook for Its Relief.—H. W. Wright.
New York Med. Jour., 1917, **105,** 788.

St. Vitus' Dance, Heredity in.—C. W. Burr.
Jour. Nerv. and Ment. Dis., 1917, **45,** 237.

Subnormal Child—Survey of School Population in Locust Point District of Baltimore.—C. M. Campbell.
Mental Hygiene, 1917, **1,** 1.

GENITO-URINARY SYSTEM

Hydrocele in Infants.—G. Caronia.
Pediatria, Naples, 1917, **25,** 212.

Nephritis in Childhood, Hypertension in.—A Study of Ninety-Three Cases.—H. K. Berkley and J. M. Lee.
Am. Jour. Dis. Child., 1917, **13,** 354.

Pyelitis in Children.—C. R. Hannah.
Texas State Jour. Med., 1917, **12,** 465.

Vulvovaginitis, Bacteriology of the Urine in Children with. Report of Two Cases of Gonococcus Cystitis.—A. B. Schwartz.
Am. Jour. Dis. Child., 1917, **13,** 420.

OSSEOUS SYSTEM

Osteitis of Tip of Petrous Portion of Temporal Bone.—A. Jouty.
Presse méd., 1917, **25,** 76.

Osteochondritis Deformans Juvenilis.—E. Jelks.
Florida Med. Assn. Jour., 1917, **3,** 257.

Superior Radio-Ulnar Synostosis; Two Cases of.—D. M. Greig.
Edinburgh Med. Jour., 1917, **18,** 281.

SKIN AND APPENDAGES

Favus Not Due to Achorion Schönleini.—D. Muĳis.
Nederl. Tidjschr. v. Geneesk., 1917, **1,** 449.

Leprosy in Argentine Province.—E. F. Solari.
Semana méd., 1917, **24,** 15.

Ringworm of Scalp Cured with Roentgen Rays; Report of Eighty-Three Cases.—F. C. Knowles and J. B. Ludy.
Pennsylvania Med. Jour., 1917, **20,** 336.

Scalp, Microbian Infections of.—Gougerot.
Progrès méd., 1917, **32,** 101.

Sweat, Blackish and Malodorous, in a Girl of Fourteen.—J. Comby.
Arch. de méd. d. enf., 1917, **20,** 147.

Trichophyton Scalp Disease in Children.—J. Comby.
Arch. de méd. d. enf., 1917, **20,** 149.

Warts, to Cure.—W. A. Münch.
Cor.-Bl. f. schweiz. Aertze, 1917, **47,** 244.

EYE, EAR, NOSE AND THROAT

Adenoids, Gritting Teeth as a Sign of.—C. E. Benjamins.
Nederl. Tijdschr. v. Geneesk., 1917, **1,** 444.

Arcus Corneae Juvenilis Due to Fatty Degeneration of the Cornea.—K. Kusama.
Mitt. a. d. Fakultät. d. k. Univ., Tokyo, 1917, **17,** 159.

Eye Diseases and Other Disturbances in Infants from Deficiency of Fat in the Food.—E. Bloch.
Ugesk. f. Laeger, 1917, **79,** 349.

Middle Ear and Mastoid, Early Care of Suppurative Conditions of.—N. S. Weinberger.
Pennsylvania Med. Jour., 1917, **20,** 321.

Otitis Media, Suppurating; Why It Becomes Chronic.
Progrès méd., 1917, **32,** 85.

Otology, Retrospect of.—M. Yearsley.
Brit. Jour. Child. Dis., 1917, **14,** 47.

Pneumococcus Infection, Pseudomembranous, in Nose and Throat.—T. Reh.
Arch. de méd. d. enf., 1917, **20,** 131.

Scarlatinal Mastoiditis, with Arrosion of Sinus Wall and Fatal Spontaneous Hemorrhages.—A. Hæggström.
Hygiea, Stockholm, 1917, **79,** 58.

Septic Sore Throat: Epidemiologic Study of a Milk-Borne Epidemic at Galesville, Wis., Due to the Use of Infected Milk.—G. W. Henika and I. F. Thompson.
Jour. Am. Med. Assn., 1917, **68,** 1307.

Septic Sore Throat Due to Milk, an Epidemic of.—E. C. Rosenow and C. L. v. Hess.
Jour. Am. Med. Assn., 1917, **68,** 1305.

Throat, Necessity of Examining, of Every Sick Child.—A. DeBlasi.
Gazz. d. osp., 1917, **38,** 301.

Tonsils and Adenoids, Eighteen Months' Observation on Mental and Physical State of Children Following Removal of.—C. J. Bloom.
New Orleans Med. and Surg. Jour., 1917, **69,** 695.

Tonsil, Faucial, and Its Relation to Systemic Conditions.—A. C. Magruder.
Colorado Med., 1917, **14,** 68.

Tonsils.—C. G. Coakley.
New Jersey Med. Soc. Jour., 1917, **14,** 138.

Tonsils and Adenoids.—J. P. McCann.
Missouri State Med. Assn. Jour., 1917, **14,** 164.

Tonsil and Adenoid Clinic.—M. M. Cullom.
Southern Med. Jour., 1917, **10,** 344.

Tonsil, Circumcision of.—F. G. Murphy.
New York Med. Jour., 1917, **105,** 785.

Tonsillar Infections, Constitutional Conditions, the Result of.—H. B. Harris.
Ohio State Med. Jour., 1917, **13,** 235.

Tonsil Region, Mixed Tumor of.—B. Quarrella.
Policlinico, Rome, 1917, **24,** 60.

Tonsillectomy, Edema of Glottis Following.—E. L. Myers.
Laryngoscope, 1917, **27,** 98.

Tonsillectomy Under General Anesthesia, New Instrument for Attachment to Suction Apparatus to Maintain Dry Field in, and to Lessen Occurrence of Postoperative Pneumonia.—S. Israel.
Laryngoscope, 1917, **27,** 102.

Tonsillectomy, Technic on.—J. Matthews.
Journal-Lancet, 1917, **37,** 190.

SURGERY AND ORTHOPEDICS

Clavicle, Fractured, Immobilization of, by Fastening Hand on Opposite Shoulder.—G. Onano.
Gazz. d. osp., 1917, **38,** 133.

Clubfoot, Modification of Usual Operation, by Addition of Tendon Transplantation or Tendon Fixation, or Both.—P. Le Breton.
New York State Jour. Med., 1917, **17,** 115.

Crippled Child.—F. C. Test.
Illinois Med. Jour., 1917, **31,** 340.

Facial Deformities, Correction of.—D. F. Aguilar.
Siglo med., 1917, **64,** 138.

Feet, Congenital Twisted, Treatment of.—E. M. Cavazzutti.
Semana méd., **23,** 645.

Flat-Foot, Use of Massage and Exercises in Treatment of.—H. A. Oldenberg.
Illinois Med. Jour., 1917, **31,** 324.

Poliomyelitis, Anterior, Orthopedic Treatment of.—E. W. Ryerson.
Illinois Med. Jour., 1917, **31,** 299.

Scoliosis.—E. G. Abbott.
Am. Jour. Orthop. Surg., 1917, **15,** 172.

Scoliosis, Treatment of.—E. G. Abbott.
Am. Jour. Orthop. Surg., 1917, **15,** 243.

THERAPEUTICS

Heliotherapy in Brazil.—A. Moncorvo, Jr.
Brazil-med., 1917, **31,** 44 and 74.

Heliotherapy, Roentgen Treatment as an Adjuvant to.—E. Amstad.
Cor.-Bl. f. schweiz Aertze, 1917, **47,** 335.

Heliotherapy in Treatment of Tuberculosis.—A. G. Shortle.
Southwestern Med., 1917, **1,** 30.

Heliotherapy in Brazil.—Moncorvo, Jr.
Brazil-med., 1917, **31,** 91.

MISCELLANEOUS

Lumbar Puncture, Anxiety About.—L. Newmark and W. F. Beerman.
Med. Rec., New York, 1917, **91,** 723.

Pediatrics and Its Relation to the General Practitioner.—W. R. Ramsey.
Wisconsin Med. Jour., 1917, **15,** 397.

Perlèche; Its Bacteriology, Symptoms and Treatment in Two Hundred and Twenty-Three Cases.—A. L. Smith.
Arch. Pediat., 1917, **34,** 274.

School Nurse.—M. A. Baker.
Journal-Lancet, 1917, **37,** 224.

Teeth, Filling Children's.—E. Zawels.
Semana méd., 1917, **24,** 139.

INDEX TO VOLUME XIII

PAGE

Abt, I. A.: Familial icterus of new-born infants 231
Acetonuria, development of, factor of inanition in, in children. B. S. Veeder and M. R. Johnston 89
Acidosis in children, with reference to pathologic anatomy. E. Lackner and H. Gauss 209
Acidosis, occurrence of, with severe diarrhea. O. M. Schloss and R. E. Stetson 218
Acrocephaly. Philip Lewin 61
Alkali and earth alkali equilibrium in spasmophilia. C. G. Grulee 44
Amyotonia congenita: A study of the known cases with a report of three new cases. H. K. Faber 305
Anatomy, physiology and pathology of childhood, recent work in. J. B. Holmes 174
Ash, R. L.: A contribution to the diagnosis of spinal cord tumors in children 487
Atrophic infants, the nonprotein nitrogen of the blood in. L. D. Minsk and L. W. Sauer 397
Berkley, H. K.: Hypertension in nephritis in childhood, with a study of ninety-three cases 354
Bernhard, A.: Studies in chemistry of the spinal fluid of children 34
Blood, the nonprotein nitrogen of, in atrophic infants. L. D. Minsk and L. W. Sauer 397
Bloom, C. J.: Hematology of infectious diarrhea 128
Brown, A.: Use of the longitudinal sinus for diagnostic and therapeutic purposes 501
Calcium metabolism in hemophilia. C. H. Laws and D. M. Cowie 236
Cerebrospinal fluid in poliomyelitis, with special reference to Lange reaction. P. C. Jeans and M. R. Johnston 239
Courtney, A. M.: Excretion of sodium chlorid solution injected subcutaneously 73
Cowie, D. M.: Calcium metabolism in case of hemophilia 236
Cyanosis, congenital, affecting all children in a family, and new method of diagnosis of congenital heart lesions, three cases of. J. H. Hess and R. G. Pearce 1
Diabetes mellitus in children. Report of two cases with study of urinary nitrogen. P. C. Jeans 145
Diarrhea, infectious, hematology of. C. J. Bloom 128
Diarrhea, severe, occurrence of acidosis with. O. M. Schloss and R. E. Stetson 218
Dubois abscesses, syphilitic disease of thymus in infants, and mode of origin of. J. Oliver 158
Dysentery, bacillary, the epidemiology of. W. G. Smillie 337
Eckles, C. H.: Gestation versus stage of lactation as factors causing abnormal composition of cow's milk 413
Endocarditis, congenital, report of a case of, with acute vegetative inflammation of the pulmonary artery and valve. H. E. Tuley and J. W. Moore 426

PAGE

Faber, H. K.: Amyotonia congenita: A study of the known cases, with a report of three new cases 305
Fales, H. L.: Excretion of sodium chlorid solution injected subcutaneously 73
Gamble, J. L.: Review of recent literature on conditions of abnormal metabolism in infants 362
Gauss, H.: Acidosis in children, with reference to pathologic anatomy.. 209
Gere, B.: Occlusion of the aqueduct of Sylvius in relation to internal hydrocephalus 461
Gestation versus stage of lactation as factors causing abnormal composition of cow's milk. L. S. Palmer and C. H. Eckles 413
Gittings, J. C.: Review of the literature of the past five years on gonococcus vulvovaginitis in childhood 438
Grulee, C. G.: Alkali and earth alkali equilibrium in spasmophilia 44
Hansell, H. F.: Review of ophthalmology 516
Heart, congenital atresia of right auriculoventricular orifice with complete absence of tricuspid valves. J. H. Hess 167
Heart lesions, congenital, three cases of congenital cyanosis affecting all children in a family, and a new method of diagnosis of. J. H. Hess and R. G. Pearce 1
Hemophilia, calcium metabolism in. C. H. Laws and D. M. Cowie 236
Hernia, diaphragmatic, case of. T. C. McCleave 252
Hess, A. F.: Infantile scurvy. IV. The therapeutic value of yeast and of wheat embryo 98
Hess, J. H.: Congenital atresia of the right auriculoventricular orifice with complete absence of tricuspid valves 167
Hess, J. H.: Three cases of congenital cyanosis affecting all children in a family and a new method of diagnosis of congenital heart lesions 1
Holmes, J. B.: Recent work in anatomy, physiology and pathology of childhood 174
Holt, L. E.: Excretion of sodium chlorid solution injected subcutaneously 73
Hydrocephalus, internal, occlusion of the aqueduct of Sylvius in relation to. M. G. Schlapp and B. Gere 461
Hypertension in nephritis in childhood, with a study of ninety-three cases. H. K. Berkley and J. M. Lee 354
Icterus, familial, of new-born infants. I. A. Abt 231
Inanition, factor of, in the development of acetonuria in children. B. S. Veeder and M. R. Johnston 89
Intestinal flora, experimental studies of. W. R. Sisson 117
Jeans, P. C.: Cerebrospinal fluid in poliomyelitis, with special reference to the Lange reaction 239
Jeans, P. C.: Diabetes mellitus in children. Report of two cases with study of urinary nitrogen 145
Johnston, M. R.: Cerebrospinal fluid in poliomyelitis, with special reference to Lange reaction 239
Johnston, M. R.: Factor of inanition in development of acetonuria in children 89
Johnston, M. R.: The nitrogen partition in the urine of normal children.. 404
Kasahara, Michio: Specific diagnosis of tuberculous meningitis. A preliminary communication 141
Lackner, E.: Acidosis in children, with reference to pathologic anatomy.. 209
Lange reaction, cerebrospinal fluid in poliomyelitis, with special reference to. P. C. Jeans and M. R. Johnston 239

PAGE

Laws, C. H.: Calcium metabolism in hemophilia 236

Lee, J. M.: Hypertension in nephritis in childhood, with a study of ninety-three cases 354

Leopold, J. S.: Studies in chemistry of spinal fluid of children 34

Lewin, P.: Acrocephaly 61

Longitudinal sinus, use of, for diagnostic and therapeutic purposes. A. Brown and G. E. Smith 501

McCleave, T. C.: A case of diaphragmatic hernia 252

Measles, Schick test in poliomyelitis, scarlet fever, in normal children and. A. Zingher 247

Meningitis, posterior (walled off); report of a case with recovery. A. Sophian 514

Meningitis, tuberculous, specific diagnosis of. A preliminary communication. Michio Kasahara 141

Metabolism, abnormal, in infants, review of recent literature on conditions of. J. L. Gamble 362

Michael, May: Résumé of work on tuberculosis in children for 1916 256

Milk, cow's, gestation versus stage of lactation as factors causing abnormal composition of. L. S. Palmer and C. H. Eckles 413

Minsk, L. D., and Sauer, L. W.: The nonprotein nitrogen of the blood in atrophic infants 397

Mitchell, A. G.: Review of the literature of the past five years on gonococcus vulvovaginitis in childhood 438

Moore, C. U.: Phenol excretion in urine of infants, including the new-born 15

Moore, J. W., and Tuley, H. E.: Report of a case of congenital endocarditis with acute vegetative inflammation of the pulmonary artery and valve 426

Nephritis, hypertension in, in childhood, with a study of ninety-three cases. H. K. Berkley and J. M. Lee 354

New-born infant, physiology of. F. B. Talbot 495

Nitrogen partition in the urine of normal children. M. R. Johnston and B. S. Veeder 404

Oliver, J.: Syphilitic disease of thymus in infants and mode of origin of Dubois abscesses 158

Ophthalmology, review of. H. F. Hansell 516

Palmer, L. S.: Gestation versus stage of lactation as factors causing abnormal composition of cow's milk 413

Paralysis, infantile, prodromal symptoms of, based on a study of 400 histories of patients admitted to the Willard Parker Hospital from July 1 to Sept. 1, 1916, inclusive. May G. Wilson 508

Pearce, R. G.: Three cases of congenital cyanosis affecting all children in a family and a new method of diagnosis of congenital heart lesions .. 1

Pediatric literature, current 110, 203, 296, 390, 457, 523

Pediatrics, progress in 110, 174, 256, 362, 438, 516

Phenol excretion in urine of infants, including the new-born. C. U. Moore 15

Physiology of new-born infant. F. B. Talbot 495

Poliomyelitis, cerebrospinal fluid in, with special reference to Lange reaction. P. C. Jeans and M. R. Johnston 239

Poliomyelitis, Schick test in scarlet fever, measles, in normal children. A. Zingher 247

Sauer, L. W., and Minsk, L. D.: The nonprotein nitrogen of the blood in atrophic infants 397

PAGE

Scarlet fever, Schick test in poliomyelitis, measles and, in normal children. A. Zingher 247

Schick test in poliomyelitis, scarlet fever, measles, and in normal children. A. Zingher 247

Schlapp, Max G.: Occlusion of the aqueduct of Sylvius in relation to internal hydrocephalus 461

Schloss, O. M.: Occurrence of acidosis with severe diarrhea 218

Schwartz, A. B.: The bacteriology of the urine in children with vulvovaginitis 420

Sisson, W. R.: Experimental studies of intestinal flora 117

Scurvy, infantile. IV. The therapeutic value of yeast and of wheat embryo. A. F. Hess 98

Smillie, W. G.: The epidemiology of bacillary dysentery 337

Smith, G. E.: Use of the longitudinal sinus for diagnostic and therapeutic purposes 501

Sodium chlorid solution injected subcutaneously, excretion of. L. E. Holt, A. M. Courtney and H. L. Fales 73

Sophian, A.: Posterior basic (walled-off) meningitis. Report of a case with recovery 514

Spasmophilia, alkali and earth alkali equilibrium in. C. G. Grulee 44

Spinal cord tumors, a contribution to the diagnosis of. R. L. Ash 487

Spinal fluid, studies in chemistry of, of children. J. S. Leopold and A. Bernhard 34

Stetson, R. E.: Occurrence of acidosis with severe diarrhea 218

Talbot, F. B.: Physiology of the new-born infant 495

Thymus, syphilitic disease of, in infant, and mode of origin of Dubois abscesses. J. Oliver 158

Tuberculosis, résumé of work on, in children for 1916. May Michael 256

Tuley, H. W., and Moore, J. W.: Report of a case of congenital endocarditis with vegetative inflammation of the pulmonary artery and valve 426

Tumors, spinal cord, a contribution to the diagnosis of, in children. R. L. Ash 487

Urinary nitrogen, diabetes mellitus in children. Report of two cases with study of. P. C. Jeans 145

Urine of infants, including the new-born, phenol excretion in. C. U. Moore 15

Urine of normal children, the nitrogen partition in. M. R. Johnston and B. S. Veeder 404

Urine, the bacteriology of, in children with vulvovaginitis. A. B. Schwartz 420

Veeder, B. S.: The nitrogen partition in the urine of normal children 404

Veeder, B. S.: Factor of inanition in development of acetonuria in children 89

Vulvovaginitis, gonococcus, in childhood, review of the literature of the past five years on. J. C. Gittings and A. G. Mitchell 438

Vulvovaginitis, the bacteriology of the urine in children with. A. B. Schwartz 420

Wilson, May G.: The prodromal symptoms of infantile paralysis based on a study of 400 histories of patients admitted to the Willard Parker Hospital from July 1 to Sept. 1, 1916, inclusive 506

Yeast and wheat embryo, therapeutic value of. Infantile scurvy. IV. A. F. Hess 98

Zingher, A.: Schick test in poliomyelitis, scarlet fever, measles and in normal children 247

CONTENTS OF VOLUME XIII

APRIL, 1917. NUMBER 4

PAGE

Amyotonia Congenita: A Study of the Known Cases with a Report of Three New Cases. H. K. Faber, M.D., San Francisco 305

The Epidemiology of Bacillary Dysentery. W. G. Smillie, M.D., Boston 337

Hypertension in Nephritis in Childhood, with a Study of Ninety-Three Cases. H. K. Berkley, M.D., San Francisco, and J. M. Lee, M.D., Nashville, Tenn 354

Progress in Pediatrics:

Review of Recent Literature on Conditions of Abnormal Metabolism in Infants. J. L. Gamble, M.D., Baltimore 362

Current Pediatric Literature 390

MAY, 1917. NUMBER 5

PAGE

The Nonprotein Nitrogen of the Blood in Atrophic Infants. L. D. Minsk, M.D., and L. W. Sauer, M.D., Chicago 397

The Nitrogen Partition in the Urine of Normal Children. M. R. Johnston, M.D., and B. S. Veeder, M.D., St. Louis 404

Gestation Versus State of Lactation as Factors Causing Abnormal Composition of Cow's Milk. L. S. Palmer, Ph.D., and C. H. Eckles, D.Sc., Columbia, Mo 413

The Bacteriology of the Urine in Children with Vulvovaginitis. A. B. Schwartz, M.D., Milwaukee, Wis 420

Clinical Department:

Report of a Case of Congenital Endocarditis, with Acute Vegetative Inflammation of the Pulmonary Artery and Valve. H. E. Tuley, M.D., and J. W. Moore, M.D., Louisville, Ky 426

Progress in Pediatrics:

Review of the Literature of the Past Five Years on Gonococcus Vulvovaginitis in Childhood. J. C. Gittings, M.D., and A. G. Mitchell, M.D., Philadelphia 438

Current Pediatric Literature 457

JUNE, 1917. NUMBER 6

PAGE

Occlusion of the Aqueduct of Sylvius in Relation to Internal Hydrocephalus. Prof. Max. G. Schlapp, M.D., and Belden Gere, M.D., New York 461

A Contribution to the Diagnosis of Spinal Cord Tumors in Childhood. Rachel L. Ash, M.D., San Francisco 487

Physiology of the New-Born Infant. Fritz B. Talbot, M.D., Boston .. 495

The Use of the Longitudinal Sinus for Diagnostic and Therapeutic Measures in Infancy. Alan Brown, M.B., and George E. Smith, B.A., M.B., Toronto, Canada 501

The Prodromal Symptoms of Infantile Paralysis Based on a Study of 400 Histories of Patients Admitted to the Willard Parker Hospital from July 1 to Sept. 1, 1916, Inclusive. May G. Wilson, M.D., New York 506

Posterior Basic (Walled-Off) Meningitis. Report of a Case with Recovery. Abraham Sophian, M.D., Kansas City, Mo 514

Progress in Pediatrics:

Review of Ophthalmology. Howard F. Hansell, M.D., Philadelphia 516

Current Pediatric Literature 523

Index to Volume XIII 531

CONTENTS

PAGE

Occlusion of the Aqueduct of Sylvius in Relation to Internal Hydrocephalus. Prof. Max. G. Schlapp, M.D., and Belden Gere, M.D., New York 461

A Contribution to the Diagnosis of Spinal Cord Tumors in Childhood. Rachel L. Ash, M.D., San Francisco 487

Physiology of the New-Born Infant. Fritz B. Talbot, M.D., Boston 495

The Use of the Longitudinal Sinus for Diagnostic and Therapeutic Measures in Infancy. Alan Brown, M.B., and George E. Smith, B.A., M.B., Toronto, Canada 501

The Prodromal Symptoms of Infantile Paralysis Based on a Study of 400 Histories of Patients Admitted to the Willard Parker Hospital from July 1 to Sept. 1, 1916, Inclusive. May G. Wilson, M.D., New York 506

Posterior Basic (Walled-Off) Meningitis. Report of a Case with Recovery. Abraham Sophian, M.D., Kansas City, Mo. 514

Progress in Pediatrics:

Review of Ophthalmology. Howard F. Hansell, M.D., Philadelphia 516

Current Pediatric Literature 523

Index to Volume XIII 531

The American Journal of Diseases of Children is issued monthly. Two volumes are published annually.

Manuscript offered for publication, books for review, and correspondence relating to the editorial management of the American Journal of Diseases of Children should be sent to Dr. Frank S. Churchill, 1259 North State Street, Chicago. Communications regarding subscriptions, reprints, etc., should be addressed to the American Journal of Diseases of Children, 535 North Dearborn Street, Chicago.

Footnotes and bibliographies should conform to the style of the Index Medicus or to that of the Index-Catalogue of the Surgeon General's Library. It is specially requested that the titles of articles and the exact date of issues, year, volume and page, be given in each case. Contributors are requested not to furnish bibliographies which are unnecessary, or mere clerical compilations. On receipt of 10 cents the American Medical Association will send a pamphlet entitled "Suggestions to Medical Authors," containing suggestions to authors on the preparation of manuscripts, rules for footnotes and bibliographies and a list of several hundred medical periodicals of the world, with approved abbreviations of each.

Authors will receive one hundred reprints free; additional copies may be obtained at cost.

The domestic subscription per year of two volumes is $3.00; Canadian rate $3.20; foreign rate $3.35 (14 shillings, 14 marks, 16 francs), including postage. Single copies 30c postpaid.

Make checks, etc., payable to the AMERICAN MEDICAL ASSOCIATION.